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E. FALKNER HILL T. CECIL GRAY

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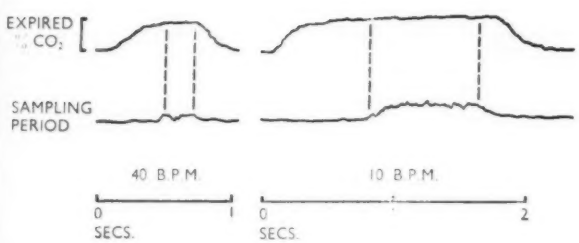
TIME-PHASED END-TIDAL CO_2 SAMPLING



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Joint Editors:

E. FALKNER HILL,
M.D., Ch.B.(Vict.), F.F.A.R.C.S., D.P.H.(Man.)

T. CECIL GRAY,
M.D., F.F.A.R.C.S., D.A

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"I would have everie man write what he knowes and no more."—MONTAIGNE

BRITISH JOURNAL OF ANAESTHESIA

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EDITORIAL

MOST of our readers will be familiar with the names of Lister, Pasteur, Koch and Semmelweis, but unless they have powerful imaginations and the leisure to apply them they will fail to grasp the emotional tones of the contemporary scene and therefore of their significance for us. The book *The Century of the Surgeon* reviewed on page 48 of this number brings this scene vividly before our eyes and reminds us of the terrible suffering that our very recent ancestors were sometimes called upon to endure. It is hard to realize that little more than one hundred years ago, the agonies of intestinal obstruction, of peritonitis, went unrelieved till death put an end to them. It is in the main to the work of these men and others whose names are less well known that we are in better case today. Our pains are rapidly relieved, our lives prolonged, and our passage through life is much less of a *via dolorosa* than it was for many of our forebears.

What did these men do? Semmelweis practised antisepsis by washing his hands in chlorine water and so reduced the appalling mortality due to puerperal fever which occurred in the lying-in hospitals all over the continent of Europe to less than 1 per cent. Pasteur discovered that fermentation was due to living organisms, Koch the cocci of sepsis, and Lister the application of antisepsis to the treatment of wounds. All alike were bitterly opposed not by scientific argument but by hostile feeling on the part of "the powers that be". Looked at from this distance the century was a supreme example of the saying "The truth shall make you free". Anaesthetics have freed man from the fear of pain, antisepsis from the fear of surgery. This is the truth that comes from knowledge. An old and renowned Scotch professor said to me, in answer to the question "Had I missed much by not learning Greek?": "Greek is just one of the avenues to Truth".

We have seen the avenue of the chemist and the bio-chemist but here is another. This work which has done so much for humanity has been done by men of different nationalities, the good is universal. It is strongly suggestive of the human brotherhood. Is the brotherhood of mankind, merely a sentimental phrase or one of wishful thinking, or is it the expression of a spiritual reality, a fundamental law of the world we live in; inexorable in its force whether we recognize it or not? If we act in accordance with it, well-being and happiness follow; against it, misery and disaster result. It, the truth of it, is our only road to freedom from the hydrogen bomb.

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RESPIRATORY EFFECTS OF THE THIOBARBITURATES*

BY

MARK SWERDLOW

Salford Royal Hospital, Manchester

IT is well known that, in clinical dosage, the thiobarbiturates depress the respiration. Considerable information is now accumulating about the mechanism and degree of this depression, but the subject is as yet scantily reported in the textbooks. The present paper reports a clinical investigation of the action of thiopentone, thialbarbitone and thiamylal on the respiration, and reviews current concepts of the respiratory effects of the thiobarbiturates in man.

MATERIAL AND METHOD

The respiratory effects of these drugs have been studied under clinical anaesthetic conditions, i.e. in premedicated patients receiving nitrous oxide–oxygen as well as thiobarbiturate. The effects of surgical stimulation have been excluded by completing the measurements before the commencement of surgery. The patients have been exclusively in the 18-50 age group and in good general condition. The drugs were injected intravenously at a standard speed in standard dose per body weight.

In pilot experiments it was found that 8 mg/kg body weight of thiopentone injected in 45 seconds gave a not too prolonged and not too severe depression of the respiration, and that with nitrous oxide and oxygen (3:1) patients would remain asleep and measurements could be made for at least 15 minutes. Some time after this dosage was adopted Dundee (1956) reported that the mean induction dose of thiopentone used in Liverpool was 8.3 mg/kg, so that the present dosage seems a reasonable one to have investigated.

The majority of previous workers considered that the equipotent ratio of thialbarbitone and thiopentone was 2:1. In the light of the present study of these drugs this ratio would seem to be reasonably accurate. With regard to equipotency between thiamylal and thiopentone there appears to be less agreement. Dornette and Tuohy (1951) and Dundee and Riding (1955) considered thiamylal to be about $1\frac{1}{3}$ more potent than thiopentone, whereas Tovell and his colleagues (1955), Mushin et al. (1955) and others could not find a significant difference in potency between the drugs. Stephen and Martin (1951) and Lund (1951) believe that thiamylal is slightly more potent than thiopentone.

In the present work the ratio 1.33:1 was first adopted and therefore the induction dose of thiamylal was 6 mg/kg and the supplementary dose 1.5 mg/kg. Seventeen patients were given this dosage of thiamylal. Of these, 2 woke up completely within 3 minutes, 1 within 6 minutes and 1 within 9 minutes. The remaining 13 received a second dose but 3 of them were so light that the stimulus of the second injection caused them to resist and the experiment was abandoned. It therefore appeared that this standard of equipotency was incorrect and in all subsequent experiments the ratio 1:1 was used, i.e. 8 mg/kg thiamylal.

In an earlier study the respiratory depression produced by equivalent doses of thiopentone and thialbarbitone was compared in two groups of 60 patients (Swerdlow, 1957). In the present investigation comparable measurements were made in 60 patients who received thiamylal sodium under similar experimental conditions. Thialbarbitone was used in 10 per cent solution; thiopentone and thiamylal in 5 per cent solution.

EXPERIMENTAL TECHINIQUE

Patients were premedicated with morphine 8-11 mg (according to size) and atropine 0.64 mg given subcutaneously 45 minutes before testing began. On arrival in the anaesthetic room the patient's mouth and pharynx were sprayed with local analgesic in order to facilitate the introduction of

* Based on a paper delivered in Liverpool on April 11, 1957, to a joint meeting of the Liverpool and Manchester Anaesthetic Societies.

TABLE I
Distribution of Patients.

	Thio-pentone series	Thial-barbitone series	Thiamylal series
No. of patients	60	60	60
Mean age (yrs.)	32.3	32.7	35.6
Mean weight (kg)	59.8	61.9	62.1
Min. weight (kg)	41	44	40
Max. weight (kg)	77	81	84
No. of males	28	26	27
No. of females	32	34	33

an oral airway later. Control readings of the respiratory rate and minute volume were then taken with the patient breathing air. The prepared dose of thiobarbiturate was then administered intravenously, a stopwatch being started synchronously with the start of the injection. The drug was injected at a steady speed, taking 45 seconds for the total dose. An oral (Guedel) airway was then inserted, a mask applied to the face and nitrous oxide and oxygen (3:1) administered via a closed circuit with intentional leak. A Bennett spirometer was inserted into the expiratory side of the Coxeter-Mushin absorber.

RESULTS

"*Apnoea Time.*"

Fifty-five to seventy seconds after zero there was usually an apnoea (with all three drugs) preceded by a few deeper breaths (fig. 1). This apnoea lasted for a variable number of seconds and upon recommencement of respiration the depth rapidly augmented. The duration of the apnoea resulting from the three drugs was measured (see table II). Under the present experimental conditions it is not always easy to note the moment of cessation of respiration so that in this work the term "apnoea time" is used to indicate the time from "zero" (i.e. the moment of commencement of injection) to the moment of recommencement of breathing. The respiration was not assisted during this period. The adoption of this measure of the duration of apnoea may involve the effects of certain variables such as circulation time and speed of action of the individual drugs. Nevertheless, it is considered that the results provide valid and useful data. Figure 2

TABLE II
Apnoea Times.

Drug	No. of cases	Number with no apnoea	Mean apnoea time of remainder
Thialbarbitone	60	18	94 ± 2.2*
Thiamylal	60	13	101 ± 2.9
Thiopentone	60	2	103 ± 2.2

* Standard error

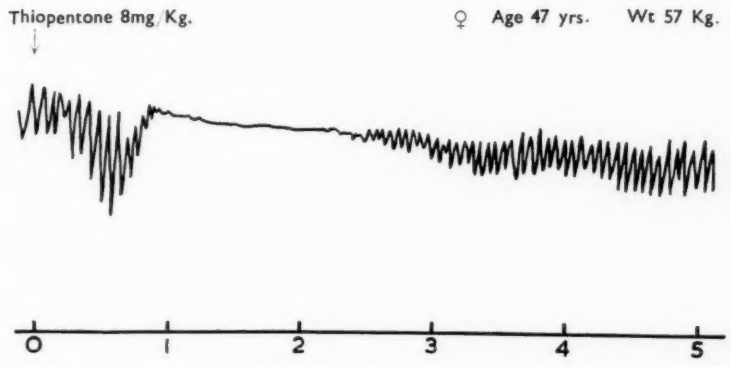

FIG. 1
Respiratory tracing.

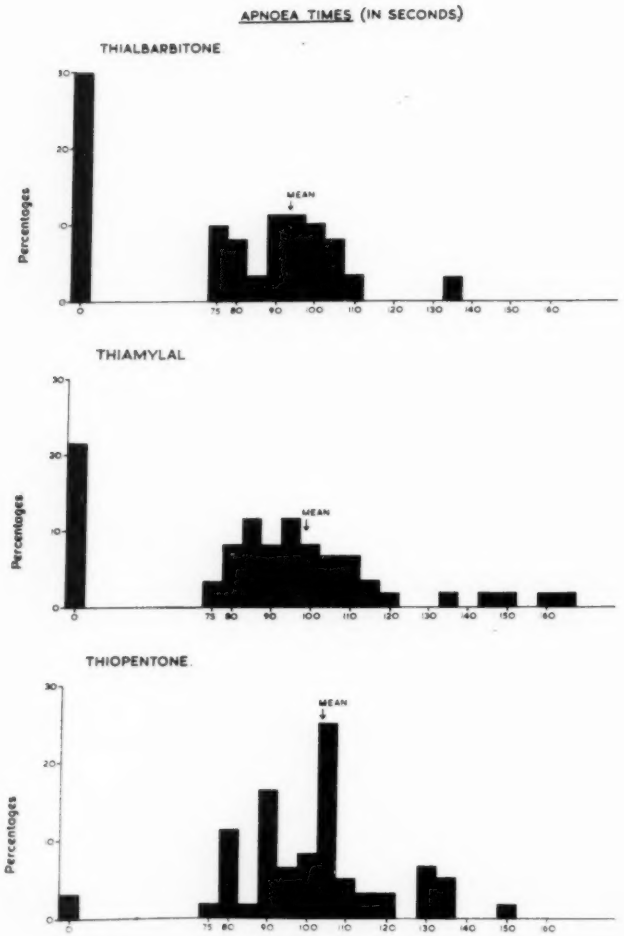

FIG. 2
Histogram of apnoea times.

shows a histogram of the apnoea times. Taking first the proportion with no apnoea, statistical test shows that both thialbarbitone and thiamylal are significantly* better than thiopentone. The difference between the former two is not significant. Taking the mean apnoea times of the remainder, thialbarbitone has a significantly lower mean than either of the other two thiobarbiturates. Combining the two criteria (i.e. proportion with no apnoea and mean apnoea time of remainder) thialbarbitone is significantly better than thiamylal which is significantly better than thiopentone. Inspection of the histogram shows that thiamylal gives more variable results than thialbarbitone and that the latter has a significantly smaller proportion of high apnoea times. There was no positive correlation between apnoea time and sex, age or weight with any of the drugs.

* The minimal level of statistical significance throughout this work is $P<0.05$.

It might be mentioned that of the 17 patients who received 6 mg/kg thiamylal 7 had no apnoea. The mean apnoea time of the remaining 10 cases was 95 seconds.

Other Respiratory Measurements.

The apnoea time having been noted, the respiratory rate and minute volume were measured 3, 5 and 9 minutes after zero. To study the effects of a supplementary dose of thiobarbiturate a second injection (¼ size of first) was given at the end of the 9-minute measurements, i.e. at 0 + 10 min. Further readings of rate and depth of respiration were taken 2, 3 and 5 minutes after this second dose. The results for thiopentone and thialbarbitone have already been reported in detail (Swerdlow, 1957) and are given here only in brief for comparison with thiamylal (see tables III to VI).

The three groups of patients who received respectively thialbarbitone, thiamylal and thiopentone are from closely similar populations (see table I).

TABLE III
Respiratory rate per min (logarithmic averages)

	Effect of 1st injection (180 patients)			Effect of 2nd injection (90 patients)		
Time:	3 min	5 min	9 min	9 min	13 min	15 min
Thialbarbitone	16.4	16.8	16.1	16.4	16.6	16.3
Thiopentone	17.9	18.0	17.5	18.3	17.3	17.1
Thiamylal	15.5	15.4	14.6	14.7	14.5	14.1
Ratio $\frac{\text{Thialbarbitone}}{\text{Thiopentone}}$	0.92	0.93	0.92	0.90	0.96	0.96
Ratio $\frac{\text{Thiamylal}}{\text{Thiopentone}}$	0.87	0.86	0.83	0.80	0.84	0.82
Ratio $\frac{\text{Thiamylal}}{\text{Thialbarbitone}}$	0.95	0.92	0.91	0.90	0.87	0.87
	Average standard error 3.1%			Average standard error 4.4%		

TABLE IV
Minute volume (ml) (logarithmic averages)

	Effect of 1st injection (180 patients)			Effect of 2nd injection (90 patients)		
Time:	3 min	5 min	9 min	9 min	13 min	15 min
Thialbarbitone	4623	5813	5806	5897	5290	5676
Thiopentone	4658	5708	5705	5597	4566	4935
Thiamylal	4421	5110	5380	5145	4681	4981
Ratio $\frac{\text{Thialbarbitone}}{\text{Thiopentone}}$	0.99	1.02	1.02	1.05	1.16	1.15
Ratio $\frac{\text{Thiamylal}}{\text{Thiopentone}}$	0.95	0.90	0.94	0.92	1.03	1.01
Ratio $\frac{\text{Thiamylal}}{\text{Thialbarbitone}}$	0.96	0.88	0.93	0.87	0.88	0.88
	Average standard error 4.4%			Average standard error 6.4%		

TABLE V
Percentage Change in Respiratory volume.

	Effect of 1st dose				Effect of 2nd dose			
	% change	3/5 min	% change	3/9 min	% change	9/13 min	% change	9/15 min
	M.V.	T.V.	M.V.	T.V.	M.V.	T.V.	M.V.	T.V.
Thialbarbitone	+25.7	+23.1	+25.6	+28.4	−10.3	−12.0	− 3.7	− 3.6
Thiopentone	+22.5	+21.5	+22.4	+28.0	−18.4	−21.0	−11.8	−13.0
Thiamylal	+15.6	+16.5	+21.7	+28.4	− 9.0	− 7.7	− 0.32	+ 0.86

It will be seen that the general pattern of the respiration following the induction and supplementary doses of thiamylal is similar to that following these doses of the other two drugs. Comparing the effects of the induction dose (see table V) the minute volume of ventilation 3 minutes after injection is very similar with all three drugs. The respiration rate, however, is significantly slower with thiamylal than with thiopentone. Allowing for the wide standard deviation the recovery at 5 and 9 minutes with thiamylal is not dissimilar from that of the other two drugs. The tidal volume changes following the initial dose are similar with all three drugs (see table VI).

Comparing the effects of the supplementary dose (table V) it will be seen that at both 3 and 5 minutes after this second dose the minute volume changes are similar for thiamylal and thialbarbitone, and both are significantly better than thiopentone; the same applies to the changes in tidal volume. The respiratory rate changes are less marked, but the supplementary dose of thiopentone produced a significantly greater fall in rate than did that of thialbarbitone.

Effect at Longer Time Intervals.

In 20 patients under the same conditions as above the standard induction dose of thiopentone was given followed by nitrous oxide and oxygen and readings were continued for 15 minutes. Table VII shows the mean changes. There appears to be an upward trend in the later values of rate and volume but owing to the large standard error in this type of work this change is not significant.

Effect of Repeated Doses of Thiobarbiturates.

It would obviously be of great interest to know the respiratory changes which follow repeated doses of thiobarbiturates. This is a much more difficult problem to tackle, involving as it does

TABLE VI
Mean Tidal Volumes (ml).

	Effect of 1st injection (180 patients)			Effect of 2nd injection (90 patients)		
Time:	3 min	5 min	9 min	9 min	13 min	15 min
Thialbarbitone	281	346	361	359	320	348
Thiopentone	261	317	334	320	265	290
Thiamylal	285	332	368	350	323	353

TABLE VII
Effect of 8 mg/kg Thiopentone (mean of 20 cases).

	3 min	5 min	9 min	11 min	13 min	15 min*
Resp. rate per min	20.9	20.2	19.1	18.4	18.5	18.5
M.V. (ml)	4644	5222	5175	5230	5241	5390
T.V. (ml)	222	254	271	284	283	291

* There were only 15 readings at this time interval.

the retention of the patient in the anaesthetic room for a long period and also the administration of perhaps a larger dose than he would otherwise have had. The numbers are therefore small; ten experiments have been done with thiopentone and ten with thialbarbitone. The experiment was started as in the " one supplementary dose " cases above. At the end of the 15-minute reading a second injection of $\frac{1}{4}$ the induction dose was given and readings of rate and minute volume were taken 2, 3 and 5 minutes after. A third dose of $\frac{1}{4}$ induction dose was then given and further readings taken 2, 3 and 5 minutes later. The results are seen in table VIII.

who received thiopentone the third and fourth doses produced apnoea lasting 2-3 minutes. It will also be noted that the mean minute alveolar ventilation immediately after the supplementary doses is very low. In some individual patients the tidal volume actually fell below the assumed dead space volume. Briscoe and his co-workers (1954) have shown that breaths of less than the known anatomical dead space can be effective in delivering inspired gases to the alveoli due to laminar flow, these gases passing as a cone down the centre of the airway. Dripps and Severinghaus (1955) therefore consider that with small tidal volumes the minute alveolar ventilation cannot be

TABLE VIII

Effect of Repeated Doses of Thiopentone and Thialbarbitone.

		S ↓			Q ↓			Q ↓			Q ↓		
	Time	3 min	5 min	9 min	12 min	13 min	15 min	18 min	19 min	21 min	24 min	25 min	27 min
THIALBARBITONE	Rate per min	16.0	16.4	15.7	15.5	15.4	15.6	15.4	15.4	15.5	15.2	15.2	15.5
	M.V. (ml)	3514	4396	4347	4012	4261	4326	3712	3877	4488	3684	3987	4421
	T.V. (ml)	220	268	277	259	277	277	241	252	290	242	262	285
	M.A.V. (ml)	624	1427	1507	1209	1478	1498	924	1093	1690	927	1237	1612
THIOPENTONE	Rate per min	17.0	17.0	16.0	16.4	16.0	15.7	15.4	15.6	15.5	15.6	15.6	15.6
	M.V. (ml)	3510	4505	4702	3659	4067	4346	2904	3709	4136	2788	3681	4216
	T.V. (ml)	206	265	294	223	254	277	189	238	267	179	236	270
	M.A.V. (ml)	476	1479	1856	738	1216	1554	169	936	1380	15.6	905	1435

S = Standard dose injected. Q = $\frac{1}{4}$ standard dose injected M.A.V. = Minute alveolar ventilation.

As regards the respiratory rate there is a slight trend towards slowing; this does not differ appreciably for the two drugs. Two minutes after each of the three supplementary doses the percentage fall in minute volume is significantly greater for thiopentone than for thialbarbitone. Three minutes after the first supplementary dose the difference is still significant but 3 minutes after the other doses there is not a significant difference. By 5 minutes after each dose there has been good recovery. It will be noted that with thialbarbitone at the end of the experiment the M.V., T.V., and M.A.V. have returned to very near the 9-minute values; after thiopentone the figures are still below the 9-minute values. In one patient calculated by subtraction of the anatomical dead space.

Effect of Nitrous Oxide.

The respiratory changes found are not, of course, entirely due to the thiobarbiturate. Apart from the premedication one must also consider the effects of the nitrous oxide and oxygen. Organe and Broad (1938) and others have commented on the " stimulating " effect of nitrous oxide and oxygen with thiopentone, an effect which appears to depend on a high percentage of nitrous oxide in the mixture, for Barton and colleagues (1946) were unable to demonstrate an increase in depth when a mixture of equal proportions of the two gases was administered.

To examine this point 25 patients, selected, premedicated and with topical preparation of the pharynx as above, were given 8 mg/kg thiopentone at the standard speed, and measurements of respiratory rate and volume were taken for 10 minutes, the patients breathing room air throughout. Of these 25 patients, 5 were very light or actually awake at 0 + 5 minutes. The data obtained in the remaining 20 patients (mean age 36.6 years; mean weight 60.8 kg) are shown in table IX, but 3 were so light at 0 + 9 minutes that a valid reading could not be taken at this time.

TABLE IX
Effects of 8 mg/kg Thiopentone in Patients Breathing Air.

Time:	Control	3 min	5 min	9 min
Number of patients	19	20	20	17
Mean R.R./min	15.9	16.9	15.9	14.8
Mean M.V. (ml)	5151	4226	4196	4136
Mean T.V. (ml)	324	250	264	279

Comparing these values with the data for thiopentone plus nitrous oxide and oxygen in tables III, IV and VI (the two sets of patients do not differ markedly in distribution of age, sex or weight) it is apparent that the initial fall in respiratory volume is similar in both series but that the patients who received thiopentone and nitrous oxide recovered to control volume by 0 + 9 minutes whereas those who received thiopentone alone did not. Statistically this apparent difference in behaviour is significant; however, the inspired P_{CO_2} of the patients breathing nitrous oxide and oxygen is likely to have been higher than that of the patients breathing air.

DISCUSSION

Investigation of the respiratory effects of these drugs can be carried out in a number of different ways (Dripps and Severinghaus, 1955):

(1) Measurement of rate and minute volume changes following administration of the drug. This method has the advantages of simplicity and ease of application but is not entirely satisfactory because respiratory changes produced by the drugs will alter the arterial CO_2 level and thereby limit the degree of depression. However, this is what happens during anaesthesia and this method gives a measure of the volume changes which actually occur in clinical practice.

(2) Measurement of changes in volume response to stimulation of the centre with CO_2. This method, too, is not entirely satisfactory because changes in ventilation will alter the amount of inspired CO_2.

(3) Measurement of minute volume changes with the alveolar P_{CO_2} maintained at a fixed level. Thus Lambertsen (1954) maintained a constant arterial P_{CO_2} by allowing changes of expired CO_2 tension (as measured by an infrared analyzer) to cause a compensatory change in inspired CO_2. Pharmacologically this is interesting but, of course, under clinical conditions the arterial P_{CO_2} does not remain at a constant level.

(4) Measurement of changes in alveolar or arterial P_{CO_2} on the assumption that CO_2 increases after administration of the drug are due to depression of the centre. However, this also presumes that the drug does not of itself cause a metabolic acidosis.

In studying the respiratory actions of the thiobarbiturates there are many complicating factors. The effects will vary with the dose, the rate of injection, the existing blood level (in cases of repeated dosage), whether the subject breathes air, oxygen or nitrous oxide–oxygen during the measurement and the presence or absence of surgical stimulation. Another important variable is premedication. Indeed Helrich and his colleagues (1956) would have it that the depression seen after administration of the thiobarbiturates is chiefly due to the premedication. These workers carried out a study in 12 young volunteers (or patients) who had no premedication or only atropine, and in 6 who received premedication (morphine 10-15 mg or pethidine 75-100 mg plus atropine). A 0.2 per cent thiopentone drip was set up and about 500 mg thiopentone were given in 20-25 minutes. The patients breathed 100 per cent oxygen and an e.e.g level of 1-2 was maintained throughout. In the majority of subjects

the rate and minute volume were not decreased and there was no change in end-expiratory CO_2. The response to stimulation with CO_2 was reduced in 7 and increased in 5. In the 6 subjects who had received premedication the end-expiratory CO_2, the respiration rate and minute volume showed variable changes but there was a markedly diminished response to CO_2. Helrich and his colleagues (1956) conclude that the premedication was largely responsible for the respiratory depression. These patients must have been in a very light plane of anaesthesia. Furthermore there is a considerable amount of variation of response and six patients is rather a small number on which to base firm conclusions. Also the dose of thiopentone does not appear to have been related to the size of the patient. Nevertheless there is no doubt that premedication does accentuate the depressant effect of the thiobarbiturates. In the present study it was noted that the apnoea time was shorter or nonexistent in patients who received no opiate premedication and also the duration of respiratory depression and of sleep were reduced.

The earlier findings (Swerdlow, 1957) appeared to indicate that a single dose of thiopentone was no more depressant than an equivalent dose of thialbarbitone but that on repeated injection thiopentone was significantly the more depressant. This is surprising as the initial depression caused by thiopentone (as reflected in the apnoea time) is significantly greater than that caused by thialbarbitone. It seems likely that this apparent discrepancy was because no measurements were made until 3 minutes after zero; had an earlier reading been taken the difference would most probably have been evident. Certainly reference to table VIII will demonstrate that 2 minutes after the supplementary doses the difference between the two drugs is significant, whereas by 3 minutes the respiration has recovered somewhat with both thiobarbiturates and there is not a significant difference. In short the initial respiratory depression caused by a single dose of thiopentone is greater than that of thialbarbitone but within a short time removal of the drug from the bloodstream and accumulation of CO_2 unite to give similar volumes of respiration. Similarly with thiamylal where the initial depression caused would appear to be somewhere between those of thialbarbitone and thiopentone.

Numerous clinical comparative studies of thiopentone, thiamylal and thialbarbitone have now been reported. However, comparison with the present findings is rendered difficult by the varied experimental conditions under which the drugs have been investigated, and also by the fact that so often opinions of respiratory depression are based on clinical impression rather than on measurement. Furthermore the dose and rate of injection are rarely stated and the dose is commonly unrelated to the size of the patient.

Wyant and his colleagues (1956) administered twice the sleeping dose of thiopentone to 7 unpremedicated volunteers. Suxamethonium was then given, the cords sprayed, and a cuffed tube inserted. Ten to 15 minutes were then allowed for the effects of the relaxant to wear off, supplementary doses of 1 per cent thiopentone being given to maintain (usually) e.e.g. level 2. Respiratory measurements were then made and arterial blood samples taken for gas analysis. Anaesthesia was then maintained at a steady level for 30 minutes (with 1 per cent thiopentone) and further measurements then made. The patients breathed room air throughout. They found:

Rate.	Increased to 190% of control at 15 min; 165% 30 min later.
M.V.	Decreased to 75% of control at 15 min; 70% 30 min later.
T.V.	Decreased to 40% of control at 15 min; 40% 30 min later.
M.A.V.	Fell from 4300 ml to around 200 ml at 15 min and at the end of the experiment.

The deleterious effect of repeated doses will be noted.

Comparing thiopentone with thialbarbitone in animals, Carrington and Raventos (1946) found that thialbarbitone caused about half as much respiratory depression as thiopentone. Bizard et al. (1952), however, found that thialbarbitone depressed the dog's respiratory centre to CO_2 for twice as long as an equivalent dose of thiopentone. Way (1951) found that when small doses of the two drugs were injected rapidly in man thialbarbitone caused the shorter period of apnoea.

With regard to thiamylal, Tovell and his colleagues (1955) reported that the incidence and duration of induction apnoea is very similar with this drug and thiopentone. Dundee (1956) states

that because of the shorter action of thiamylal, periods of respiratory depression should not be as prolonged as with thiopentone. Wyant and his colleagues (Wyant et al., 1957; Dobkin and Wyant, 1957) have recently provided considerable information about the respiratory effects of thiamylal and thiopentone as well as of other thiobarbiturates. In the former paper (Wyant et al., 1957) they report studies carried out in 5 unpremedicated young healthy males, the thiobarbiturates being given in random order to each of the subjects. They injected a $2\frac{1}{2}$ per cent solution at a speed of 1 ml per $1\frac{1}{2}$ seconds until the patient closed his eyes and after a short pause the sleep dose was repeated at a moderately rapid speed. The average period of apnoea with twice the sleep dose of thiamylal was 29 seconds, only 2 of the patients becoming apnoeic. The average apnoea with thiopentone was 38 seconds, all becoming apnoeic. Allowing for the lack of premedication and the rather higher dosage used the results correspond well with those of the present work.

Wyant and his co-workers also state that with thiopentone in 4 of the 5 patients the apnoea was interrupted just after its commencement by a few small breaths; after thiamylal this occurred only once. (This effect was not noted in the present work where a nonrecording type of spirometer was used.) They found that with both drugs the respiration rate remained steady throughout the testing period. The minute volume and tidal volume were, after the first few minutes, similar with both drugs up to 10 minutes. After this time, however, these values remained steady with thiamylal while with thiopentone they showed a progressive rise. Possibly because of the use of nitrous oxide and oxygen and premedication and the difference of dosage these findings have not been corroborated in the present investigation.

The mechanism whereby the thiobarbiturates cause this depression of respiration has been studied by a number of workers. As long ago as 1936, Marshall and Rosenfeld noted a progressive reduction in response to CO_2 with deepening barbiturate anaesthesia in animals. In 1941-43 came the classic investigations of Moyer and Beecher (1942) and Dripps and Dumke (1943). The former workers administered 5 per cent thiopentone intravenously to dogs breathing room air. An initial dose of 25 mg/kg was given followed by intermittent doses as necessary to maintain a level depression of the spinal reflex. They found a diminishing response to CO_2 with increasing depth but little reduction in the response to hypoxia. Dripps and Dumke investigated the respiratory response to thiopentone in decerebrate, unpremedicated cats and dogs. They too found a progressively diminishing response to CO_2 with increasing depth of anaesthesia while the sensitivity of the carotid and aortic chemoreceptors was increased or unchanged.

More recently Patrick and Faulconer (1952) in clinical studies administered thiopentone either by intermittent injections of a $2\frac{1}{2}$ per cent solution or by continuous 1 per cent drip and followed the depth of anaesthesia by e.e.g. They found that the mean M.V. diminished with increasing depth of anaesthesia and that the response to stimulation with 5 per cent CO_2 also diminished with depth. The mean arterial CO_2 was only slightly raised during lighter levels of anaesthesia, suggesting that the centre still retains control by the normal stimulus although the threshold may be raised. Another interesting investigation is that of Barton, Wicks and Livingstone (1946), who maintained a steady, moderate depth of anaesthesia with intermittent injections of $2\frac{1}{2}$ per cent thiopentone and took measurements of the arterial oxygen and CO_2. They found that 10 minutes after the commencement of anaesthesia the CO_2 had risen slightly but there was no fall in arterial oxygen tension. Administration of 100 per cent oxygen and then nitrous oxide–oxygen (50:50) resulted in a rise in arterial oxygen and a slight fall in CO_2. A rise in P_{CO_2} after thiopentone was also noted by Lucas and Milne (1953) who found arterial P_{CO_2} ranging from 45 to 94 mm Hg.

From these investigations it is clear that the thiobarbiturates depress the central chemical control of respiration more than the peripheral (aortic and carotid body) reflex mechanism and that in deep anaesthesia the latter system plays a large part in the maintenance of respiration. This has an important clinical application. After a large dose of thiobarbiturate respiratory depression will not respond to carbon dioxide. Furthermore if the anaesthetist simply administers 100 per cent oxygen it will prevent the chemoreceptor stimula-

tion of breathing and CO_2 may accumulate to such a degree as to exert a depressant effect which can end in a fall in blood pressure and death.

The influence of the Hering-Breuer reflex during thiopentone anaesthesia has also been investigated. Scott and his colleagues (1948) administered thiopentone to dogs and supplied oxygen by endotracheal insufflation. They found that pressure on the chest wall would return respirations in the apnoeic animal. In the vagotomized animal rendered apnoeic with thiopentone, lying on the vagotomized side prolongs the apnoea while lying on the opposite side will increase sensitivity to the reflex or cause resumption of respiration. Bilateral vagotomy abolishes the reflex response. Whitteridge and Bulbring (1946) believe that many of the respiratory effects of various anaesthetics are due to specific changes in excitability of the pulmonary stretch receptors. Lee (1953) states that thiopentone exaggerates the Hering-Breuer reflex in direct proportion to the depth of anaesthesia. Thus pressure on the bag at the end of inspiration can produce several seconds' apnoea, a procedure which he finds useful during some radiological investigations.

McCann (1948) has made a study of thiopentone apnoea. He considers that there is during induction apnoea an increasing tonic contraction of the expiratory muscles and that after a varying time the tonus is released and normal rhythmic respiration is re-established. He believes that this is due to an initial irritative action of thiopentone either on the expiratory neurones in the respiratory centre or on the motor cells of the cortex. McCann considers that induction apnoea is different from that caused by large or repeated doses of thiopentone where there is a sustained depression of both the inspiratory and expiratory neurones of the centre.

Pharmacologically, the degree of respiratory depression will obviously be related to the blood (and hence brain) concentration of the thiobarbiturate. The profound depression which follows within a minute of the injection of the induction dose gives way to a rapidly improving ventilation as the drug is distributed throughout the tissues. The increasing depth of breathing will, of course, also reflect accumulation of CO_2 during the period of acute depression and this factor will tend to disguise the difference between the effects of different drugs, for the greater the initial depression the greater the compensatory effect of CO_2. Brodie (1952) has shown that following intravenous injection the blood level of thiobarbiturate falls steeply due to rapid distribution throughout the body fluids. The speed of this redistribution varies with the molecular structure of the particular barbiturate. This partition between plasma and tissue fluid compartments continues until equilibrium is reached. According to Goldbaum (1948) with anaesthetic doses of thiopentone such equilibrium is reached in 5–10 minutes in man.

This paper has been largely concerned with the effects of the thiobarbiturates in healthy patients of young and middle age. It is perhaps superfluous in this journal to note that excessive respiratory and other depression must be expected unless cautious dosage is employed in the aged, the poor-risk, the anaemic, the patient who has been receiving large doses of sedatives, and the patient with impaired oxygenation due to cardiac or pulmonary disease. As has been seen, such dangers will be accentuated if a multiple dose technique is used. The importance of adequately assisting the respiration immediately after each injection of thiobarbiturate is also apparent. The modern trend is towards limitation of the use of thiobarbiturates to induction only and this together with graduation of dosage in relation to the patient's condition, slow injection and perhaps the use of more dilute solutions will do much to increase the safety of this valuable group of drugs.

SUMMARY

Thiopentone, thialbarbitone and thiamylal, in equivalent dosage and under standardized clinical conditions, were administered each to 60 patients and the resulting respiratory changes were measured. Apnoea following the induction dose was significantly more common with thiopentone than with the other two drugs; the mean apnoea time was significantly lower with thialbarbitone than with thiamylal, which latter was significantly better than thiopentone. The respiratory volume changes at 3, 5 and 9 minutes after the induction dose were similar with all three drugs. A supplementary dose, given at 0+10 minutes, caused significantly greater respiratory depression in the thiopentone group than in the other two series.

Two groups of patients were given the standard induction dose followed, at equal intervals, by three supplementary doses of thiopentone and thialbarbitone respectively. The fall in respiratory volume following each of the supplementary doses was significantly greater with thiopentone.

In further experiments the respiratory effect of the standard induction dose of thiopentone was investigated with the patients breathing air. The respiratory volume, after the first few minutes, was significantly lower than in similar patients breathing nitrous oxide and oxygen.

Other researches into the nature and mechanism of the respiratory changes following the thiobarbiturates are reviewed. It is considered that these drugs depress the central control of respiration more than the peripheral mechanism and that in deep anaesthesia the latter mechanism is largely responsible for the maintenance of respiration. It is also believed that there is an increase in the Hering-Breuer reflex under thiobarbiturate anaesthesia.

The clinical implications are discussed.

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REFERENCES

Barton, G. D., Wicks, W. R., and Livingstone, H. M., (1946). The effects of Pentothal, alone and in combination with oxygen or nitrous oxide-oxygen on arterial blood gases. *Anesthesiology*, **7**, 505.

Bizard, G., Vanlerenberghe, J., and Robelet, A. (1952). Action comparée du pentothal et du kemithal sur l'excitabilité du centre respiratoire par le gaz carbonique. *C.R. Soc. Biol. (Paris)*, **146**, 1121.

Briscoe, W. A., Forster, R. E., and Comroe, J. H. (1954). Alveolar ventilation at very low tidal volumes. *J. appl. Physiol.*, **7**, 27.

Brodie, B. B. (1952). Physiological disposition and chemical fate of thiobarbiturates in the body. *Fed. Proc.*, **11**, 632.

Carrington, H. C., and Raventos, J. (1946). Kemithal: a new intravenous anaesthetic. *Brit. J. Pharmacol.*, **1**, 215.

Dobkin, A. B., and Wyant, G. M. (1957). The physiological effects of intravenous anaesthesia on man. *Canad. Anaesth. Soc. J.*, **4**, 295.

Dornette, W. H. L., and Tuohy, E. B. (1951). Clinical trial of surital sodium in 1,200 cases of general anaesthesia. *Curr. Res. Anesth.*, **30**, 159.

Dripps, R. D., and Dumke, P. R. (1943). Effects of narcotics on the balance between central and chemoreceptor control of respiration. *J. Pharmacol. exp. Ther.*, **77**, 290.

—— Severinghaus, J. W. (1955). General anaesthesia and respiration. *Physiol. Rev.*, **35**, 741.

Dundee, J. W. (1956). *Thiopentone and Other Thiobarbiturates*. Livingstone: London.

—— Riding, J. E. (1955). A clinical trial of thiamylal as an intravenous anaesthetic in 1,750 cases. *Brit. J. Anaesth.*, **27**, 381.

Goldbaum, L. R. (1948). An ultraviolet spectrophometric procedure for the determination of barbiturates. *J. Pharmacol. exp. Ther.*, **94**, 68.

Helrich, M., Eckenhoff, J. E., Jones, R. E., and Rolph, W. D. (1956). The influence of opiates on the respiratory response of man to thiopental. *Anesthesiology*, **17**, 459.

Lambertsen, C. J. (1954). *Pharmacology in Medicine*. New York: McGraw-Hill, 17. 55. (Quoted by Dripps and Severinghaus, 1955.)

Lee, J. A. (1953). *A Synopsis of Anaesthesia*, p. 25. Bristol: Wright.

Lucas, B. G. B., and Milne, E. H. (1953). The role of carbon dioxide in anaesthesia. *Proc. roy. Soc. Med.*, **46**, 365.

Lund, P. C. (1951). Surital sodium—a new intravenous anaesthetic agent. *Amer. J. Surg.*, **81**, 637.

Marshall, E. K., jr., and Rosenfeld, M. J. (1936). Depression of respiration by oxygen. *J. Pharmacol. exp. Ther.*, **57**, 437.

McCann, J. C. (1948). Pentothal need during surgical anaesthesia. *Curr. Res. Anaesth.*, **27**, 314.

Moyer, C. A., and Beecher, H. K. (1942). Effects of barbiturate anaesthesia on the integration of respiratory control mechanisms. *J. clin. Invest.*, **21**, 429.

Mushin, W. W., Henderson, A. D., and Mapleson, W. W. (1955). Comparative potencies of thiamylal and thiopentone. *Brit. J. Anaesth.*, **27**, 374.

Organe, G. S. W., and Broad, R. J. B. (1938). Pentothal with nitrous oxide and oxygen. *Lancet*, **2**, 1170.

Patrick, R. T., and Faulconer, A., jr. (1952). Respiratory studies during anaesthesia with ether and with pentothal sodium. *Anesthesiology*, **13**, 252.

Scott, J. C., Reed, E. A., Saris, D., and Ramirez, H. P. R. (1948). Role of Hering-Breuer reflex under deep pentothal anaesthesia. *Amer. J. Physiol.*, **154**, 428.

Stephen, C. R., and Martin, R. (1951). Clinical experience with surital sodium in anaesthesia. *N. C. med. J.*, **12**, 501.

Swerdlow, M. (1957). The effect on the respiration of thialbarbitone and thiopentone. *Brit. J. Anaesth.*, **29**, 21.

Tovell, R. M., Anderson, C. C., Sadove, M. S., Artusio, J. F., Papper, E. M., Coakley, C. S., Hudon, F., Smith, S. M., and Thomas, G. J. (1955). A comparative clinical and statistical study of thiopental and thiamylal in human anesthesia. *Anesthesiology*, **16**, 910.

Way, G. L. (1951). Le Kemithal. *Praxis.*, no. **14**, 283.

Whitteridge, D., and Bulbring, E. (1946). Changes in activity of pulmonary receptors in anaesthesia and their influence on respiratory behaviour. *Brit. med. Bull.*, **4**, 85.

Wyant, G. M., Kilduff, C. J., Merriman, J. E., and Dobkin, A. B. (1956). Evaluation of Dolitrone. *Canad. Anaesth. Soc. J.*, **3**, 291.

—— Dobkin, A. B., and Aasheim, G. M. (1957). Comparison of seven intravenous anesthetic agents in man. *Brit. J. Anaesth.*, **29**, 194.

Brit. J. Anaesth. (1958), **30**, 13

ENDOBRONCHIAL ANAESTHESIA WITH THE CARLENS CATHETER

BY

A. VIVIAN JENKINS AND GORDON CLARKE

Department of Anaesthetics, Royal Infirmary, Manchester

THE double-lumen catheter introduced by Carlens (1949) for bronchospirometry was first used in anaesthesia in 1950 with the primary object of preventing the spread of secretions from one lung to the other during pulmonary resection (Björk and Carlens, 1950). While this method of endobronchial anaesthesia has been the subject of several reports from other countries (Björk, Carlens and Crafoord 1952; Björk, Carlens and Friberg, 1953; Rose, 1954; Lynch and Feraru, 1957), it does not appear to have achieved any degree of popularity in this country and we feel it would be of advantage to outline our experiences of the method over the past twenty months. We have used the catheter partly to prevent the spread of secretions but also with the almost equal object of facilitating the surgeon's work in the chest.

Prior to the introduction of the catheter used in this series of cases, bronchospirometry by means of specially designed double-lumen, flexible, endobronchial catheters had been performed by Gebauer (1939). Later Zavod (1940) and Marsh (1953) described similar devices. Of these, only the catheter described by Carlens has found any application in clinical anaesthesia.

There are three main principles used in preventing the spread of secretions in thoracic surgery. An endobronchial tube with an inflatable cuff may be introduced into the bronchus of the healthy lung (Gale and Waters, 1931; Magill, 1936); or the bronchus to the affected lung or segment may be separated by means of a blocker (Archibald, 1935; Magill, 1936; Halton, 1943; Moody et al., 1947), or by tamponade (Crafoord, 1938); or the patient may be placed in the prone position. (Overholt, Langer, Szypulski and Wilson, 1946; Brown, 1948). All these methods are attended by certain disadvantages. With an endobronchial tube, it is difficult to aspirate secretions from the diseased lung and impossible to inflate or deflate it during the operation. Endobronchial blockers are notoriously difficult to retain in the correct position and again it is impossible to inflate the lung during the operation. In addition it may be impossible to deflate the lung, thereby making the surgery more difficult.

The prone position is unsatisfactory because the alternate dissection from the front and back of the hilum of the lung which so much facilitates surgery is not possible. Again the affected lung cannot be inflated and deflated independently at will.

In this series of cases the lateral position has been used exclusively and in all patients the catheter was introduced using the technique described by Jenkins (1956). Thiopentone anaesthesia with suxamethonium chloride as a relaxant provided a wide open immobile larynx. Topical analgesia of the respiratory tract was produced with 4 per cent lignocaine, with the aid of a Multicaine spray.

With increasing experience it has been possible to introduce the catheter in the majority of cases with almost as much ease as an ordinary endotracheal tube and we do not consider difficulty of intubation to be a major obstacle to its use.

The catheter, originally manufactured in two sizes corresponding to 35 Fr. and 39 Fr., is now available in the three sizes detailed in table I. The generally accepted principle has been adopted of introducing the largest catheter possible, without using force. In the majority of adult men the 41 Fr. size and in adult women the 39 Fr. size was used. In only five patients have we failed to introduce a catheter because the

TABLE I
Dimensions of available Carlens catheters.

Catheter size (Fr. gauge)	External diameter (mm)	Internal cross-section area (sq.cm)		Approximate equivalent Magill tube (one lumen)
		R. lumen	L. lumen	
41	15	0.44	0.45	7
39	13	0.38	0.36	5–6
37	12.5	0.31	0.33	4–5

smallest size was too big. Indeed, as experience was gained in intubation, it was found that the 41 Fr. size could frequently be inserted easily even into medium sized adults. This suggests that a still larger catheter would find a place in thoracic anaesthesia.

The prevention of transbronchial spread of secretions has been our primary indication for the employment of the catheter. It is used for this purpose whether or not the patient is producing sputum pre-operatively and has been used in thoracoplasty solely for this purpose. The majority of patients with tuberculosis today have little or no sputum because of adequate pre-operative therapy, but it is nevertheless found that manipulation of the lung during surgery may result in a considerable amount of secretion entering the bronchial tree. Furthermore, in patients with bronchial carcinoma there is often some degree of bronchial stenosis with secondary suppuration distal to it. Purulent material from this may easily flood the bronchial tree. The catheter was also used with good effect in one patient who had copious haemoptysis from a bronchial neoplasm pre-operatively; and it will, of course, prevent contamination of the contra-lateral bronchial tree as a result of bleeding occurring during the operation, either before or after the bronchus is opened.

Secondly, we have found the use of the catheter to be of invaluable assistance to the surgeon during the operation. Indeed some of the operations would have been extremely difficult and even impossible without it. It has already been mentioned that the lateral position can be used with impunity, thus facilitating access to the chest and dissection of the hilum. The lung can be deflated at will, thereby increasing the operating space. The bronchus may be divided and left open at any suitable time during the operation. It is unnecessary to apply a clamp to any part of the bronchus which is to be conserved, a procedure which impairs the blood supply to the bronchus and has been held to be a cause of post-operative bronchial fistula. Resection of a lobe together with part of the main bronchus, followed by anastomosis of the remaining lobar bronchus to the main bronchus, or to the trachea (Thomas, 1956), has been greatly facilitated.

After the bronchus has been closed during a resection or after an anastomosis has been performed, it is possible to reinflate the lung to test for leakage and then, if necessary, deflate it while further suture is carried out. This, of course, cannot be done once a blocker has been removed or an endobronchial tube withdrawn into the trachea.

In patients with bronchopleural fistulae the use of the tube constitutes a satisfactory method of preventing spill of secretion and leak of gases through the fistula (Parkhouse, 1957). In the presence of a large fistula it would be essential, of couse, to introduce the catheter under topical analgesia with the patient conscious, as for bronchospirometry, and thereafter use one-lung anaesthesia. However, no such cases are included in the present series.

For these reasons the use of the Carlens catheter has become routine at this hospital and has been accepted unanimously by the surgeons as an important advance in thoracic surgery.

During the twenty months covered by this survey the authors have used the catheter in 330 cases classified as shown in table II. During the same period other anaesthetists in this hospital have intubated a further 460 patients with the tube, and we have been privileged to use the total experience gained as the basis for our report.

TABLE II
Operations in which Carlens catheter has been used.

Operation	No. of cases
Pneumonectomy	73
Lobectomy	108
Segmental resection	66
Thoracoplasty	43
Decortication	14
Bronchopleural fistula	2
Non-pulmonary	7
Thoracotomy (inoperable)	17
Total	330

Before anaesthetizing the patient it is essential to test the catheter carefully. The integrity of both cuffs is tested by inflation under water and careful inspection of the sites at which the cuffs are sealed on to the tube should be made, since it is here that we have found leakage to occur most commonly. The integrity of the septum is tested by blowing down each lumen in turn, simultaneously occluding the corresponding outlet. Any leakage of air can then only occur through a defective septum. This will also demonstrate the patency of each lumen.

During intubation certain difficulties may arise. Introduction of the tip of the catheter into the glottis may be difficult, especially in patients who have a complete dentition or have one or two teeth missing from the right upper jaw. In these latter the tube tends to stick in the gaps. Intubation may also be difficult in patients with a long palate or a receding lower jaw, as indeed is ordinary endotracheal intubation. This may be overcome by the use of Magill forceps to lift the tip of the tube into the larynx.

As soon as the tip enters the larynx, the tube must be rotated to bring the hook to face anteriorly. If the tube is pushed too far into the larynx before this is done the hook catches on the right aryepiglottic fold and prevents further rotation.

A tube which is too large often enters the larynx easily but is held up at the level of the cricoid ring, this being the narrowest part of the larynx. It has been suggested that the bulk of the hook is the cause of the failure of the tube to pass through the cricoid, and further that, since its presence is not essential for accurate positioning, it may well be removed. We disagree with this. Measurement of the tube has shown that the diameter at the level of, and including, the hook is no greater than that of the tracheal portion of the tube. It is obvious, therefore, that removal of the hook will still not allow such a tube to pass.

A tube which is too big must not be forced into the trachea. Not only will damage to the larynx and trachea occur directly, but also the tube may fail to rotate back in the trachea into the correct position. We have seen this on two occasions, when the tube remained twisted in the trachea and the left bronchial tube entered the right main bronchus.

It might be thought that the insertion of such a large and irregular tube must inevitably cause some damage to the larynx and trachea. Siebecker and Land (1956), however, in a series of 206 patients found no laryngeal lesion which could be attributed to the tube.

Several methods of connecting the tube to the anaesthetic apparatus have been described (Rose, 1954; Beer, 1954; Lynch and Feraru, 1957) all of which we feel to be unnecessarily complicated. We use two Cobb's suction unions, one inserted into the proximal end of each lumen and connected by short rubber tubes to the Y-piece from a dental nasal inhaler. This in turn is connected to a Waters canister and to the anaesthetic machine. A size 4 union is used with a 41 Fr. tube and a size 3 with the other two tubes. Before the tube is introduced it must be ascertained that the plug is easily removable and gastight. A lung can be separated from the circuit by applying a clamp across the corresponding rubber tube. It can then be deflated simply by removing the plug of the Cobb's union. When necessary, bronchial suction is carried out by inserting the suction catheter through the Cobb's union and the corresponding side of the Carlens catheter. The connecting tube must first be clamped if this has not been done already. Thus there is no need to disconnect any part of the assembly other than the plug of the union.

After the tube is in place it is secured by strapping and a prop is placed between the teeth to prevent it being bitten. The cuffs are then inflated, and it is advantageous to do this while the patient is still apnoeic. The left cuff should be

inflated first. Our procedure is to clamp off the right side and remove the plug of the right Cobb union. Gentle pressure is applied to the reservoir bag while the left cuff is inflated until reflux of gases up the right side just ceases. The cuff inflation tube is then clipped. The clamp is removed and the plug replaced. Pressure is again applied to the bag while the tracheal cuff is inflated until the reflux of gases up the trachea just ceases. By this means a gastight seal of both cuffs is achieved without an excessive pressure being exerted by them on the tracheal and bronchial mucosae. Tests are now made to ensure that the tube is correctly placed. Pressure on the reservoir bag should inflate the chest, showing that the tube is in the trachea and has not slipped backwards into the oesophagus during introduction. Each side is clamped off in turn and each lung inflated in turn. The chest is both inspected and auscultated to make certain that gases are freely entering through the unclamped side and are not passing into the clamped side. As a final check a suction catheter is passed into each side to make sure that it will pass easily into both main bronchi.

During the operation inflation, deflation and suction of each lung can be carried out as already described. Certain specific problems do, however, arise.

During pneumonectomy it is desirable to close the bronchus flush with the tracheal wall. The hook of the tube on the right side, and the left bronchial tube on the left side will interfere with this surgical procedure. For a planned right pneumonectomy a tube with the hook removed before insertion is used. If the pneumonectomy is not anticipated the surgeon either amputates the hook before closing the bronchus or deflects it from the carina into the left main bronchus using a suitable forceps. It is desirable to use a hookless tube where possible, otherwise one rapidly acquires a collection with this modification!

We normally regard the presence of the hook as essential for correct positioning of the tube, and only in the special case of right pneumonectomy do we dispense with it. It has been found that even a 41 Fr. size tube without a hook can easily pass too far into the left main bronchus, and on one occasion the tip of the tube entered the left upper lobe bronchus. Thus careful testing to ascertain that the tube is in the correct position is doubly necessary when using a hookless tube.

For left pneumonectomy the problem is more difficult. There are two methods of dealing with the situation. Either the catheter may be drawn back sufficiently far to bring the whole tube into the trachea before closing the left bronchus, or the catheter may be removed entirely and a cuffed endotracheal tube substituted. In either case the bronchial tree must be cleared of blood and secretions before disturbing the tube. The cuffs must be deflated before moving the tube and, if it is to remain in the trachea, only the tracheal cuff is reinflated in the new position. Partial withdrawal of the catheter is not a very satisfactory solution to the problem for several reasons. In the first place the catheter tends to migrate down again into the surgical field to the annoyance of the surgeon, while the anaesthetist, as he withdraws it farther and farther, is constantly worried by the possibility of its suddenly coming completely out of the glottis. In one instance this did occur and serious hypoxia resulted before an endotracheal tube could be inserted. Secondly, measurement of the tube shows that the distance between the tip and the proximal end of the tracheal cuff is about 11 cm, which is equal to the length of the trachea. Thus, when withdrawn sufficiently far, there is a strong possibility that the tracheal cuff will lie in the larynx and be re-inflated there. This we feel to be undesirable. Thirdly, when the catheter lies in the trachea the tip of the left endobronchial segment presses firmly against the left wall of the trachea, and this may cause severe mucosal injury. For these reasons, therefore, the authors favour substituting a cuffed endotracheal tube at the stage of bronchial transection and closure. This is not so difficult and hazardous as might be thought, but it must be done quickly to avoid hypoxia. Before removing the catheter the patient is well ventilated with oxygen for one minute and, if necessary, given a further small dose of thiopentone and/or relaxant. The laryngoscope should be inserted and the glottis visualized before the catheter is removed. If there was difficulty in intubating the patient during induction, the alternative procedure of withdrawing the catheter into the trachea may be employed. During the bronchial closure the patient is inflated intermit-

tently, the surgeon occluding the bronchial opening when necessary.

The only absolute contra-indication to use of a Carlens tube is stenosis of the left main bronchus, but the tube does have certain disadvantages. The major criticism has been the resistance to gas flow consequent upon the relatively small diameter of each lumen. It has been shown (Gaensler, Maloney and Björk, 1952) that the resistance to dry air afforded by the 39 Fr. size tube was equivalent to 7 cm water on the right side and 5 cm on the left at a flow rate of 30 metres per minute. This is about four times the resistance of the normal human respiratory tract. In a similar study (Swenson and Birath, 1957) it was shown that the flow through the catheter during clinical use must be turbulent, because the gases passing through it are saturated with water vapour. In this case the resistance is proportional to the square of the flow rate and inversely proportional to the fifth power of the radius of the tube. This shows the important effect that small changes in diameter have upon the resistance of the tube, and emphasizes the importance of using the largest possible tube. It can be calculated that the resistance of the 39 Fr. size is about half as much again as the 41 Fr. size, the difference in lumen diameter being only 0.5 mm and the resistance of the 37 Fr. size is twice that of the 41 Fr. size, the difference in lumen diameter being 1 mm. These workers have also shown the superiority of all three sizes of Carlens catheter over the Marsh and Zavod catheters which were tested in a similar way.

While there is undoubtedly some increased resistance to respiration when a Carlens catheter is used, this can be compensated for by increased pressure on the reservoir bag when controlled respiration is employed during thoracic anaesthesia. Even during spontaneous respiration—which we allow for a short time during positioning and, of course, during bronchospirometry—we have not noticed any dyspnoea or "resistance breathing".

In the emphysematous patient, however, the increased resistance to deflation of the lung does assume considerable importance, and in patients with gross emphysema the catheter is probably contra-indicated.

SUMMARY

The Carlens double-lumen catheter has been used in anaesthetizing 330 patients for thoracic surgery. The clinical use of the catheter is described.

Indications for the use of the catheter, its superiority over other methods and certain problems arising during anaesthesia are discussed.

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REFERENCES

Archibald, E. (1935). A consideration of the dangers of lobectomy. *J. thorac. Surg.*, **4**, 335.

Beer, E. G. (1954). Connector for endobronchial anaesthesia. *Anesthesiology*, **15**, 106.

Björk, V. O., and Carlens, E. (1950). The prevention of spread during pulmonary resection by the use of a double-lumen catheter. *J. thorac. Surg.*, **20**, 151.

—— —— Crafoord, C. (1952). The open closure of the bronchus and the resection of the carina and of the tracheal wall. *J. thorac. Surg.*, **23**, 419.

—— —— Friberg, O. (1953). Endobronchial anaesthesia. *Anesthesiology*, **14**, 60.

Brown, A. I. P. (1948). Posture in thoracic surgery. *Thorax*, **3**, 161.

Carlens, E. (1949). A new flexible double-lumen catheter for bronchospirometry. *J. thorac. Surg.*, **18**, 742.

Crafoord, C. (1938). On the technique of pneumonectomy in man. *Acta chir. scand.*, **81**, 1 (Suppl. 54).

Gaensler, E. A., Maloney, J. V., and Björk, V. O. (1952). Bronchospirometry. II: Experimental observations and theoretical considerations of resistance breathing. *J. Lab. clin. Med.*, **39**, 935.

Gale, J. W., and Waters, R. M. (1931). Closed endobronchial anaesthesia in thoracic surgery. *J. thorac. Surg.*, **1**, 432.

Gebauer, P. W. (1939). A catheter for bronchospirometry. *J. thorac. Surg.*, **8**, 674.

Halton, J. (1943). A method of controlling bronchial secretion in thoracic surgery. *Lancet*, **1**, 12.

Jenkins, A. V. (1956). Carlens catheter: a simple method of intubation. *Brit. J. Anaesth.*, **28**, 85.

Lynch, C. G., and Feraru, F. (1957). Endobronchial anesthesia. *Anesthesiology*, **18**, 138.

Magill, I. W. (1936). Anaesthetics in thoracic surgery with special reference to lobectomy. *Proc. roy. Soc. Med.*, **29**, 643.

Marsh, K. (1953). A double-lumen catheter for bronchospirometry. *J. thorac. Surg.*, **25**, 495.

Moody, J. D., Trent, J. C., and Newton, G. W. (1947). An endobronchial balloon for control of bronchial secretions during lobectomy and pneumonectomy. *J. thorac. Surg.*, **16**, 258.

Overholt, R. H., Langer, L., Szypulski, J. T., and Wilson, N. J. (1946). Pulmonary resection in the treatment of tuberculosis. *J. thorac. Surg.*, **15**, 384.

Parkhouse, J. (1957). Anaesthetic aspects of bronchial fistula. *Brit. J. Anaesth.*, **29**, 217.

Rose, C. E. (1954). Anaesthesia in thoracic surgery for tuberculosis with special reference to the use of the Carlens catheter. *Curr. Res. Anaesth.*, **33**, 297.

Siebecker, K. L., and Land, J. F. (1956). Lack of laryngeal trauma following the use of Carlens catheter. *Anesthesiology*, **17**, 660.

Swenson, E. W., and Birath, G. (1957). Airflow resistance in bronchospirometric catheters. *J. thorac. Surg.*, **33**, 275.

Thomas, C. P. (1956). Conservative resection of the bronchial tree. *J. roy. Coll. Surg. Edinb.*, **1**, 169.

Zavod, W. A. (1940). Bronchospirometry. I: Description of the catheter and technique of intubation. *J. thorac. Surg.*, **10**, 27.

BOOK REVIEW

Introduction to Anesthesia. By R. D. Dripps, M.D., J. E. Eckenhoff, M.D., and L. D. Vandam, M.D. Published by W. B. Saunders Company, Philadelphia and London (1957). Pp. 266; illustrated. Price 33*s*.

The authors inform us that this book is a descendant of a smaller work, privately printed and circulated in 1949 and again, on revision, in 1953. It is indeed fortunate that this latest edition has become available to the widest circle of anaesthetists, and especially to those who are responsible for the instruction of trainees in the specialty. The intention to include the sort of information that is regularly passed on to juniors by word of mouth during operating sessions has been fulfilled. Equally the authors have avoided unnecessary detail in describing various techniques but have concentrated upon fundamental principles. These emphasize on repeated occasions that the object of the book is well described in its subsidiary title, "The Principles of Safe Practice." All junior anaesthetists will find this book a valuable addition to the first shelf of their library—it would form a suitable modern companion to Guedel's classic monograph on "Inhalation Anesthesia." Senior anaesthetists will be gratified to find that many of their views are so firmly supported and so clearly expressed. As this is an introductory volume, many of the specialized aspects of anaesthesia have been omitted, e.g., hypothermia and hypotension, but each chapter carries at its close a short list of references for guidance to wider reading.

It is reasonable in a book intended as a basis for fundamental and safe teaching of anaesthetics to draw attention to a few statements which could be revised. Speaking of the fundamentals of muscle relaxants, the text states that following the use of d-tubocurarine and the waning of its effect, the subsequent injection of decamethonium or succinylcholine in the usual paralyzing doses is relatively ineffective. Many cases of prolonged apnoea have in fact followed just such a procedure.

During the induction of anaesthesia described in Chapter 14 it is recommended that a 5 litres per minute flow rate for the first ten to fifteen minutes may be reduced to 2–3 litres per minute after this. So low a flow rate would encourage a student to ignore the accumulation of carbon dioxide. The same chapter deals with intravenous barbiturate anaesthesia and the danger of intra-arterial injection, but it fails to indicate the advisability of confining injections of thiopentone to areas free from arteries or nerves, a vital instruction to the junior anaesthetist. Finally, when describing the technique of lumbar puncture, the following sentence appears: "The anaesthetist's hands should have been washed and dried but need not be surgically clean." Even the following insistence on a "no touch" technique hardly justifies this statement.

It is probably unfair to pick out such minor blemishes when there is so much to praise and admire, but they seem of some importance. The line drawings are clear and refreshing in a textbook; the text is agreeable and pleasant to read and there is great value in the novel arrangement of the contents. These carry the subject in sequence from the pre-anaesthetic medication to the postoperative period. Well printed and indexed and attractively bound, this is a book which all anaesthetists should be glad to possess.

H. H. Pinkerton

Brit. J. Anaesth. (1958), **30**, 19

A CASE OF POSTOPERATIVE RESPIRATORY INSUFFICIENCY AND PROLONGED UNCONSCIOUSNESS

BY

HILARY L. MORSS AND THOMAS W. BAILLIE

Department of Anaesthesia, Stobhill Hospital, Glasgow, Scotland

IN 1955, Rukes et al. described the dramatic response of several cases of pre- and postoperative shock to the administration of intravenous hydrocortisone where standard methods of treatment by blood transfusion and vasopressor agents had failed.

Our interest in the possible value of hydrocortisone in the treatment of some cases of postoperative respiratory insufficiency was roused by Hügin's (1957) analysis of Hunter's (1956) cases of "neostigmine-resistant curarization" especially by his observation that some of these patients presented a clinical picture suggestive of acute suprarenal cortical insufficiency. His own words on the subject are: "It seems to me possible that these patients could be saved by the rapid infusion of hydrocortisone, and there is probably no connection between this complication and the use of relaxant drugs or neostigmine".

As such cases fortunately occur infrequently in any one anaesthetist's practice, it is perhaps of interest to record a recent one of this type in which intravenous hydrocortisone formed part of the therapy.

CASE REPORT

A 78-year-old man was admitted to hospital at 2.15 p.m. on 27.5.57 with a history of severe lower abdominal pain of 24 hours duration. There was no history of vomiting, constipation or diarrhoea. Among the positive findings were bilateral reducible inguinal herniae and generalized abdominal tenderness, most marked in the right lower quadrant. Temperature 100.2°F. Pulse rate 110/minute. Respiratory rate 24/minute. B.P. 145/90 mm mercury. Urine: reaction acid with a trace of albumin.

He had previously had operations for repair of inguinal herniae in 1935 and 1945 and an operation for intestinal obstruction in 1947.

After examining the patient at 3 p.m., the surgeon decided that laparotomy should be performed and ordered morphine sulphate 10 mg subcutaneously. The patient was seen 2 hours later by the anaesthetist who considered him to be remarkably alert mentally for his age of 78. He was a big, robust man with a slightly toxic appearance, but with no apparent dehydration. Pre-operative medication of atropine sulphate 0.6 mg subcutaneously was ordered.

At 6 p.m. laparotomy was performed and the findings were:

(1) Generalized purulent peritoneal exudate.
(2) Paralytic distension of small and large intestines.
(3) Gangrenous perforated appendix.
(4) Incomplete obstruction of ileum by an omental band.
(5) Loop of ileum in right inguinal hernial sac.

The operation consisted of appendicectomy and division of the omental band.

Anaesthetic Procedure.

Anaesthesia was induced with thiopentone sodium 300 mg given slowly intravenously, followed by d-tubocurarine chloride 20 mg. After rhythmic inflation of the lungs for two minutes, intubation was attempted unsuccessfully due to coughing and movement of the patient's head and arms. Following a supplementary intravenous injection of 200 mg thiopentone sodium, he settled and intubation with a no. 10 cuffed endotracheal tube was performed with ease.

Anaesthesia was maintained with nitrous oxide 3 litres and oxygen 1½ litres employing a Waters' to-and-fro rebreathing unit with controlled leak, and assisted respiration. Within 10 minutes of induction of anaesthesia the patient was breathing again with vigour, and in order to facilitate exploration of the abdomen a further 10 mg d-tubocurarine and 25 mg pethidine hydrochloride were given. Before closure of the peritoneum, 5 mg d-tubocurarine was administered.

Resuscitation.

At the conclusion of the operation, the administration of neostigmine methyl sulphate 2.5 mg, preceded by atropine sulphate 0.6 mg, resulted in regular respiration of adequate amplitude, but the patient remained unconscious. Throughout operation his condition had remained good, blood pressure and pulse maintaining their pre-operative values. Blood loss had been negligible.

On his return to the ward at 7.30 p.m. the hitherto adequate tidal exchange gave place to an inadequate diaphragmatic respiration with tracheal tug, whose response to intravenous injections of atropine and neostigmine in the previous dosage was short lived.

Re-intubation was performed with ease and with no reaction other than an occasional extrasystole over the next few minutes. There was no response either to inflation with 100 per cent oxygen or to inflation with 6 per cent carbon dioxide in oxygen. Intermittent positive pressure respiration with CO_2 absorption was then re-instituted. An intravenous infusion of 5 per cent dextrose in saline was set up. A short period of deep thoraco-abdominal respiration followed the administration of 4 ml 25 per cent solution of nikethamide but there was no alteration in depth of consciousness. Since 500 mg thiopentone is a very large dose in the aged, bemegride 25 mg was administered intravenously, but this failed to alter the amplitude of respiration or the level of consciousness. The patient remained deeply comatose with pupils small, but not pin point and unresponsive to direct light.

At 11.15 p.m. an intravenous solution of hydrocortisone 100 mg in 540 ml dextrose 5 per cent in water was run in rapidly and towards the end of this infusion the tone of the jaw muscles increased markedly and the tracheal tug disappeared. Respiration increased in amplitude to such a degree that assistance was rendered unnecessary. Moreover for the first time respiration became mainly thoracic in type.

Despite the fact that the improvement in respiration was maintained the systolic blood pressure fell 1 hour later to 90 mm mercury. 15 mg methylamphetamine sulphate was given intravenously followed by 15 mg intramuscularly with no immediate response. Two hours later the systolic blood pressure was still 90 mm mercury but respiration had become more shallow. The administration of nikethamide in the previous dosage resulted in a similar increase in amplitude of respiration for about 2 minutes. From this point onwards, his tidal volume and blood pressure remained unchanged, but the level of consciousness gradually lightened until at 8.30 a.m. the patient would no longer tolerate the endotracheal tube. At 10 a.m. he was fully conscious and able to speak clearly and intelligently.

Blood Chemistry.

Records of serum electrolytes are not available for the pre-operative period but the results obtained on the first postoperative day are as follows:

Serum urea	91 mg per cent.
Serum chlorides	96 m.equiv/l.
Serum alkali reserve	24.8 m.equiv/l.
Serum potassium	6.2 m.equiv./l.

DISCUSSION

Over the past year several anaesthetists have reported the occurrence of postoperative respiratory insufficiency, generally in elderly and/or extremely ill patients. (Hunter, 1956; Foster, 1956; Kenny, 1956; Burchell, 1956). The dramatic improvement following intravenous hydrocortisone suggests that in this patient at least the response to anaesthesia and operation was due to a modification of suprarenal cortical activity. Foster (1956) administered 17-hydroxycorticosterone to two patients suffering from postoperative respiratory insufficiency, one of whom recovered consciousness temporarily. It would appear that the second patient, who also died, was already in a state of advanced circulatory failure when the drug was administered.

A diagnosis of adrenocortical insufficiency in such patients is not unreasonable. As a result of the probable atrophy of the suprarenal glands in old age a modification of the response to stress is not unlikely. In this case the stress was considerable with infection, haemoconcentration, electrolyte imbalance, narcosis and operation as contributory factors.

Foster (1956) after administration of potassium to several such patients with some success, concluded that the postoperative respiratory insufficiency was related to low intracellular potassium which allowed molecules of muscle relaxant to penetrate the blood/brain barrier and thus exert a central depressant action on brain cells. It has been shown however, that an increase in the potassium content of blood plasma may result in increased corticoid secretion (Vogt, 1951) and it is possible that this was partly the reason for the response of some patients in Foster's series to the intravenous administration of potassium solutions.

From the time of medication pre-operatively to that of prolonged unconsciousness postoperatively, the response of this patient to narcotics was abnormal. Two hours after the administration of 10 mg morphine subcutaneously this 78-year-old normotensive patient was very alert. A further abnormal reaction was the apparent resistance to the hypnotic action of thiopentone. In contrast, the duration and depth of unconsciousness postoperatively were not in keeping with this initial resistance. It is possible that the duration of unconsciousness may have been prolonged by the administration of dextrose solutions of which he had 1 litre of 5 per cent. Although the effect of administration of glucose on subjects under thiobarbiturate narcosis has been demonstrated only in experimental animals (Lamson, Greig and Robbins, 1949). An annotation (*Lancet*, 1950) warns of the possibility in humans.

Caughey and Garrod (1954) in a study of coma and disturbances of consciousness in hypopituitarism have noted that endocrine failure, particularly

of the suprarenal cortex, seemed to be an important factor in lowering the threshold of consciousness. It has also been noted (Dundee, 1957) that prolonged unconsciousness may occur in patients suffering from Addison's Disease in whom replacement therapy had been inadequate, particularly when such patients are already under the influence of opiates and thiopentone. Dundee has also reported cases of prolonged unconsciousness and circulatory failure following anaesthesia in cases of drug-induced adrenocortical insufficiency.

The response of the brain to narcotics depends on the rate of cerebral metabolism and this latter may be altered by the blood level of adrenocorticoids. It seems reasonable to imagine, " without penetrating ever deeper into the maze of speculative pharmacology " (Hewer, 1957), that under a condition of altered cerebral metabolism due to modification of corticoid secretion an abnormal response to narcotics may be produced.

SUMMARY

A case of postoperative respiratory insufficiency and prolonged unconsciousness is presented.

The management is described and the possible aetiological factors discussed.

ACKNOWLEDGMENTS

We should like to thank Mr. F. Smith for his permission to publish the details of the case, and Dr. O. M. Watt for his encouragement and interest.

REFERENCES

Burchell, G. B. (1956). Irreversible curarization. *Brit. J. Anaesth.*, **29**, 127.

Caughey, J. E., and Garrod, O. (1954). Coma and allied disturbances of consciousness in hypopituitarism. *Brit. med. J.*, **2**, 554.

Dundee, J. W. (1957). Anaesthesia and surgery in adrenocortical insufficiency. *Brit. J. Anaesth.*, **29**, 166.

Foster, P. A. (1956). Potassium depletion and the central action of curare. *Brit. J. Anaesth.*, **28**, 488.

Hewer, C. L. (1957). Neostigmine-resistant curarization. *Brit. med. J.*, **1**, 463.

Hügin, W. (1957). Neostigmine-resistant curarization. *Brit. med. J.*, **1**, 463.

Hunter, A. R. (1956). Neostigmine-resistant curarization. *Brit. med. J.*, **2**, 919.

Kenny, S. (1956). Neostigmine-resistant curarization. *Brit. med. J.*, **2**, 1113.

Lamson, P. D., Greig, M. E., and Robbins, B. H. (1949). Potentiating effect of glucose and its metabolic products on barbiturate anaesthesia. *Science*, **110**, 690.

Lancet. Annotation (1950). Potentiation of barbiturate anaesthesia by glucose. **1**, 820.

Rukes, J. M., Orr, R. H., Forsham, P. H., and Galante, M. (1955). Hydrocortisone: its newer analogs and Aldosterone as therapeutic agents. *Ann. N.Y. Acad. Sci.*, **61**, 448.

Vogt, M. (1951). Cortical secretion of isolated perfused adrenal. *J. Physiol.*, **113**, 129.

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CASE REPORT OF DEATH DURING ANAESTHETIC ASSOCIATED WITH ADRENAL HYPOFUNCTION

BY

H. Q. O. WHEELER

Hexham, Northumberland

February 10, 1957. The patient was admitted as an emergency. She was a somewhat obese woman aged 50 with a 3-year history of "difficulty with her bowels". For the past 9 days she had had lower abdominal pain and had 24 hours slight sickness.

Previous History.

Pleurisy and small chest operation aged 15. Laparotomy for tuberculosis of abdomen (details not available).

Examination.

Hard working, intelligent farmer's wife. Slightly obese, but stated she had lost a little weight. Abdomen: no pain now, but considerable discomfort. T. 101°C, P.R. 98/min, B.P. 125/70 mm Hg. On general examination there was nothing abnormal except slightly harsh breath sounds at the site of her old pleurisy.

Locally. ? mass in L.I.F. On rectal examination a mass was felt—fixed but not tender.

Blood Transfusion.

Four pints (2 l.) between February 7 and 11.

Anaesthetics. February 11.

(1) Thiopentone, nitrous oxide, gallamine 80 mg (registrar administrated). Drainage of pus from abscess adjoining tumour.

Progress. Slight diarrhoea—anaemia. Apyrexial and well by February 25.

(2) *March 4.* Thiopentone, cyclopropane (consultant administration).

Sigmoidoscopy and e.u.a. The mass was found to be very adherent and fixed to the uterus.

(3) *March 13.* Thiopentone, nitrous oxide, pethidine, suxamethonium drip (administered by a clinical assistant).

Laparotomy. Left iliac colostomy. Extraction of 1 tooth and two roots.

(4) *March 15.* Thiopentone, cyclopropane (consultant administration). Colostomy "tidied". Unexpected extensive vascular control required.

(5) *April 4.* Thiopentone, cyclopropane, suxamethonium (consultant administration).

Dental extractions: $\frac{\text{5rt 43 / 12345rt}}{\text{54321 / 12345rt.}}$

(6) *April 6.* Thiopentone (consultant administration). Sigmoidscopy and e.u.a.

General condition. Very well indeed. The patient was eager to get on with the major procedure—excision of rectum.

(7) *May 27.* Temperature normal, P.R. 72/min, B.P. 140/90 mm Hg, C.V.S., and R.S. clear.

Operation. Abdominoperineal resection of rectum. The tumour was mobilized more easily than expected. No secondaries palpable. Hysterectomy. Anaesthetic (consultant, self).

The following notes were made:

1452 hrs. Thiopentone 2 mg, suxamethonium 30 mg. Intubation with cuffed tube. Maintenance with nitrous oxide, oxygen, halothane.

1458 hrs. Operation commenced; d-tubocurarine 20 mg.

1505 hrs. Pulse rate 106/min. Blood pressure 110/90 mm Hg.

1515 hrs. Pulse rate 96/min. Blood pressure 110/90 mm Hg.

1535 hrs. Pulse rate 80/min. Blood pressure 105/90 mm Hg.

1545 hrs. Pulse rate 76/min. Blood pressure 100/90 mm Hg. Halothane off and cyclopropane substituted.

1555 hrs. Pulse rate 76/min. Blood pressure 100/90 mm Hg.

1605 hrs. Pulse rate 80/min. Blood pressure 110/80 mm Hg.

1615 hrs. Pulse rate 86/min. Blood pressure 120/80 mm Hg. Colour in hands and ears poor: circulation poor. Tidal volume adequate. Tube not kinked. Heart and breath sounds appear normal.

1625 hrs. Pulse rate 120/min. Blood pressure 125/80 mm Hg.

1635 hrs. Pulse rate 110/min. Blood pressure 100/85 mm Hg. Colour a little better.

1645 hrs. Pulse rate ? 100/min. Blood pressure 75/60 mm Hg. Amphetamine 2 ml intravenously.

1650 hrs. ? Not obtainable. Apparent cardiac arrest. Cardiac massage: intracardiac nikethamide 4 ml and amphetamine 2 ml. There was no response to cardiac massage and other resuscitation procedures.

Postmortem examination.

Almost complete replacement of gland tissue by carcinoma in both adrenals, confirmed histologically (old tuberculosis of lung with pleural adhesions).

It was interesting to note how well this patient remained all through her hospital stay. Her first five anaesthetics were perfectly straightforward. It was a little surprising that her e.u.a. and sigmoidoscopy on April 6 were so easily carried out on only thiopentone in divided doses up to a total of 500 mg. The suddeness of the final circulatory collapse was striking.

Brit. J. Anaesth. (1958), **30**, 23

AN ELECTRONIC TIME CYCLED RESPIRATOR

BY

JAMES ROCHFORD, RONALD F. WELCH AND DEREK P. WINKS

Department of Pathology, Barnet General Hospital, Barnet, Herts.

THE rhythmic inflation of the lungs, whether manually or by machine, is a well established procedure in anaesthesia and in the treatment of respiratory insufficiency. There is, however, a divergence of opinion as to the most suitable type of automatic respirator, and as to whether they are as efficient as or more efficient than manually controlled respiration. It must be remembered that the principles which have been established regarding cardiorespiratory effects apply equally to manual as to automatic respiration.

Much reliance is placed on the so-called "experienced" hand in manually controlled respiration. How many anaesthetists can estimate the amount of positive pressure used on inspiration, the amount of ventilation to 100 ml of gas per breath, or the length of the inspiratory and expiratory period, without the use of instruments? Maloney (1954) states that of 50 anaesthetists observed, 47 were unable adequately to estimate ventilation by manual and visual observance of the reservoir bag movement.

All types of intermittent positive pressure respiration, whether by hand or machine, impose a strain on the circulation by decreasing the pressure gradient which allows blood to flow from the great veins into the right heart. The positive pressure exerted on the lung alveoli tends to interfere with alveolar capillary flow; the higher the pressure and the longer the period over which it is exerted the more the capillary flow is occluded (Cournand et al., 1948; Motley et al., 1948).

This impairment of the circulation may be made minimal by manipulating the timing of the respiratory cycle and the pressure exerted, always subject to an adequate ventilatory volume. Providing ventilation is satisfactory the shorter the length of inspiration and the longer the length of expiration the more the heart is able to compensate for the circulatory depression due to pressure breathing.

It is evident that if pressure breathing is to be used three essential factors must be continuously and exactly measured and controlled; anything else is inspired guesswork. These factors are:

(1) The pressure changes in the lung during the cycle.
(2) The measurement of the expiratory volume.
(3) The actual time taken by inspiration and expiration.

We have been concerned with time cycled (pressure limited) respirators for four years in preference to the more popular and more available pressure and volume cycled (pressure limited) machines.

The advantage of time cycling appears to be that the timing of the cycle can be set exactly to provide for minimum depression to circulation. Reference to figure 1 shows a pressure volume diagram constructed for a patient. It will be seen that with inspiration at 1.25 sec and a pressure of 10 cm H_2O the same ventilation can be produced as with a pressure of 8 cm H_2O and an inspiratory period of 2 sec. In the first instance the total pressure period in the minute is 18.75 sec whereas in the second instance the pressure period is 30 sec, the shorter inspiratory period allowing the heart an extra 11 seconds in the minute in which to work unimpaired. Spalding (1955) demonstrated that, providing there is no obstruction to respiration, the amount of tidal air is almost proportional to the inspiratory pressure, and that a large proportion of the tidal air enters during the early part of inspiration. He showed, and our figures agree with his, that adequate tidal air (subject to a satisfactory instantaneous airflow) may be obtained by an inspiratory period of 0.6 sec and that little is to be gained by prolonging it beyond

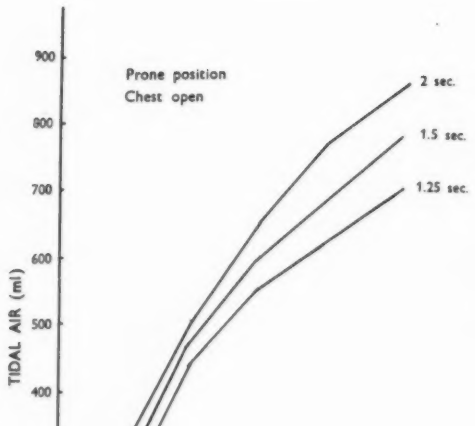

FIG. 1

At 450 ml. Ideal tidal vol.

Inspiratory pressure	Inspiratory period	Total pressure period per minute
1. 8 cm	2 sec	30 sec
2. 9 cm	1.5 sec	22.5 sec
3. 10 cm	1.25 sec	18.75 sec

1.5 sec. We have rarely used a period longer than 1.25 sec, the respiratory rate varying from 12 to 18 per minute. It is simple to compose a pressure volume diagram for each patient at different inspiratory lengths which may help in the choice of the best combination of pressure and time for the required expiratory volume. The respiratory volume was determined from the Radford (1955) nomogram corrected for anaesthetic factors. This volume was taken as a guide to the minimum volume of respiration necessary for adequate ventilation. When in doubt we have collected end expiratory gas from the trachea by means of fine polythene tubing and analyzed the gas for CO_2 percentage by means of a Draeger analyzer. This method, while not very accurate, is clinically useful.

Time cycling does not suffer from the disadvantage of pressure cycling machines where a leak in the gas circuit may lengthen the inspiratory period until such time as the leak is big enough, when the whole cycling mechanism fails, as the cycling pressure is never reached. Gordon et al. (1956) have demonstrated an advantage of the time cycled machine over the volume cycled machine, in that the shape of the inspiratory curve, curve I (time cycled), is more physiologically ideal than curve II (volume cycled) and produces less cardiac impairment.

We have constructed these machines on a one-way circuit so that it is possible to meter exact percentages of gases to the patient, which is of advantage when using nitrous oxide. The cost of this increased gas flow is repaid by savings in use of soda lime and the lessened use of analgesics. The further advantage of an open circuit is that there is no question of increased carbon dioxide due to channelling or altered absorption by the soda lime. Gaensler (1956) in an experiment demonstrated that a conscious volunteer breathing in a closed circuit system with a conventional 1 lb. canister of soda lime increased his ventilation from 6 l./min to 20 l./min in the first 45 minutes. He concludes that the ordinary 1 lb canister is reasonably efficient for 15 mins of respiration. The addition of an electronic triggering device subject to any dominant rhythm between 12–60 respirations per minute increases the scope of the machine, as it may be used when spontaneous respiration is allowed or on the conscious patient with respiratory insufficiency where it has shown itself to be comfortable.

Variants of the machine have been used by one of us in over 600 thoracic operations (table I) and more than 100 nonthoracic cases as well as a few cases of respiratory incapacity arising from disease or drug overdosage. The longest period of continuous use has been 60 hours. The ages of the patients have varied from 10 to 72 years. In no case was a negative phase used because of any deleterious effects on the cardiovascular system even though massive haemorrhage has occasionally occurred. A negative phase of

TABLE I

Pneumonectomy	100
Thoracotomy	75
Lobectomy (and lingula)	158
Segmentals and wedges	117
Diaphragmatic hernia	4
Mitral valvotomy	62
Patent ductus	4
Oesophagectomy	6
Pericardial cyst	1
Hydatid cyst	1
Retrosternal thyroid	3
Repair oesophageal fistula	2
Repair bronchial stump	4
Cysts of lung	4
Decortications and 1st stage thoracoplasty	59+
	600+

-5 to -10 cm H_2O has, however, been found extremely useful in the type of case in which expiration is prolonged and the lungs rigid.

The negative pressure has been obtained by adding a T-piece to the expiratory outlet attached to a sucker, the negative pressure being controlled by a variable orifice. The expired air has been measured by a Cowan and Parkinson dry gas meter graduated to read 1500 ml on one revolution and in the latest machine by a Wright's anemometer (to be fitted).

THE RESPIRATOR

The machine consists of a gas circuit with electromagnetically operated gas valves controlled by an electronic circuit.

(a) *Gas Circuit* (fig. 2).

Anaesthetic or other gases flow continuously under a pressure of approximately 5 lb./sq.in from a Boyle's or similar machine into a 1.5 litre rubber concertina bag; a spill valve is fitted to prevent overdistension. A large coil spring whose tension is adjusted by a manually operated worm drive gear applies pressure to the bag and thus controls the pressure within the gas circuit.

Two alternative methods have been used with success; a sliding weight on a bar attached to the top of the bag and at the other end to a fixed pivot allows equally close control of pressure, this depending on the position on the bar at which the weight is clamped. A Walton 4 Mixing Valve eliminates the need for a rubber bag altogether and permits instantaneous flow rates of up to 180 l./min but requires an input pressure of 10 lb./sq.in and cannot, therefore, be attached to a Boyle's machine without special adaptation. The pressure in the gas circuit can be varied between 5 and 30 cm of water pressure. The flow from the bag is controlled by a gas valve of piston or poppet type the stem of which is operated by the extended armature of a solenoid. From this gas valve the gases pass through wide, smooth bore, nondistensible plastic tubing to a Y junction at the anaesthetic mask or intratracheal tube adaptor, at which point a manometer is connected. Exhaled gases flow through a parallel length of plastic tubing to a second solenoid operated gas valve which opens to atmosphere but to which a negative phase device or an anemometer can be attached. Instantaneous or peak flow of the apparatus is of the order of 80 l./min which, although not attaining the ideal of 100 l./min, is efficient (Proctor, 1955).

(b) *Electronic control device* (fig. 3).

The control device is a free running transistor multivibrator. In this circuit two transistors conduct alternately, switching each other automatically, the length of time of conduction of each depending upon the value of coupling capacitors and their leak resistors. The latter are variable and can be adjusted to allow periods of conduction varying from 0.5 to 4.5 seconds. The multivibrator circuit opens and closes a relay through an output transistor and the contacts of this control the gas valves. One gas valve is thus indirectly connected to one of the two transistors of the multivibrator, the other gas valve to the remaining transistor of the pair. As each transistor conducts, its associated gas valve opens for a period governed by the setting of the leak resistance thus permitting independent control of both inspiratory and expiratory periods. The control, being by variable resistances, is infinitely variable and not by steps. The gas valves can never be both open or both closed; switching is instantaneous, positive and with a short transition period.

(c) *Trigger device.*

The gas valves have one-way valves incorporated in them and a tambour is connected into the gas circuit between the two main valves. In

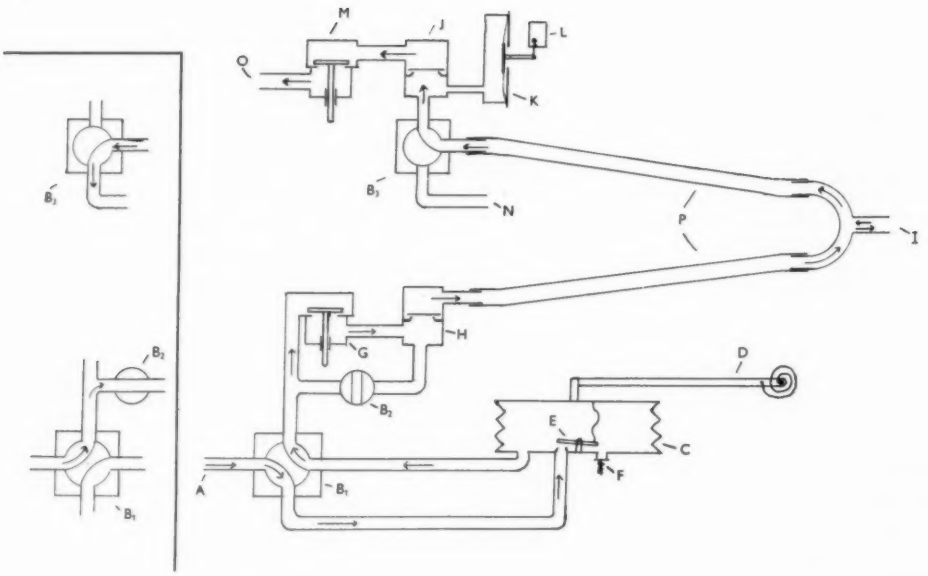

FIG. 2

A.	Gas inlet.
B_1, B_2, B_3.	Automatic/Manual control (shown in "automatic" position in main diagram: inset shows "manual" position).
C.	"Concertina" bag.
D.	Lever and spring.
E.	Shut-off valve, preventing over distension of bag.
F.	Excess pressure release valve.
G.	Solenoid operated valve controlling inspiratory phase.
H.	Nonreturn valve.
I.	Connection to face mask or endotracheal tube.
J.	Nonreturn valve.
K.	Tambour.
L.	Microswitch.
M.	Solenoid operated valve controlling expiratory phase.
N.	Connection to manual bag.
O.	Outlet to atmosphere or spirometer.
P.	Flexible tubing to patient.

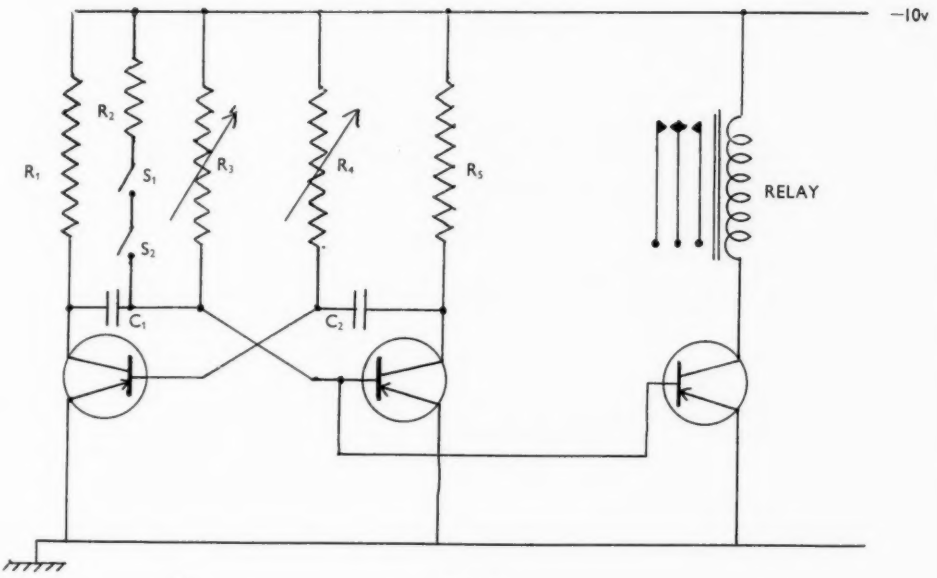

FIG. 3

R_1, R_5.	Collector load resistors.	C_1, C_2.	Coupling capacitors.
R_2.	Trigger circuit current-limiting resistor.	S_1.	Trigger microswitch.
R_3, R_4.	Variable leak resistors.	S_2	Trigger/automatic switch.

the event of the patient attempting to inspire during the expiratory phase, a negative pressure is produced as the one-way circuit valve disc closes, and the movement of the tambour diaphram operates a microswitch which short circuits the variable leak resistor controlling the inspiratory valve. The multivibrator immediately switches and opens the inspiratory valve and gas under controlled pressure inflates the lungs. The rise in gas pressure immediately opens the microswitch and the multivibrator reverts to its former rhythm. By setting the multivibrator to the longest expiratory period the patient can trigger the machine continuously and weak inspiratory efforts can be assisted.

(d) *Manual operation.*

By a single movement of a lever the machine can be switched out of the anaesthetic circuit and manual control of respiration instituted. The lever diverts the incoming gases from the concertina bag into the manual control bag held by the anaesthetist and makes direct connection between this and that part of the gas circuit between the two gas valves.

(e) *Use with explosive anaesthetics.*

Thermionic valves can be used in the electronic circuit in place of transistors, but their use not only increases the bulk but, because of the heat dissipation, prevents use of explosive anaesthetics and renders the construction of hermetically sealed units very difficult. Transistors operate at very low power and their heat dissipation is negligible. The transistor control unit is here sealed hermetically in a metal box, ceramic sealed terminals being used for electrical connections and oil filled variable resistors with sealed spindles being the only controls other than the trigger switch brought to the outside of the case. The trigger switch operates on a current of 9 mA at 10 volts d.c. in a noninductive circuit. This, the only unsealed unit, is to be considered as an essentially safe circuit as defined in the

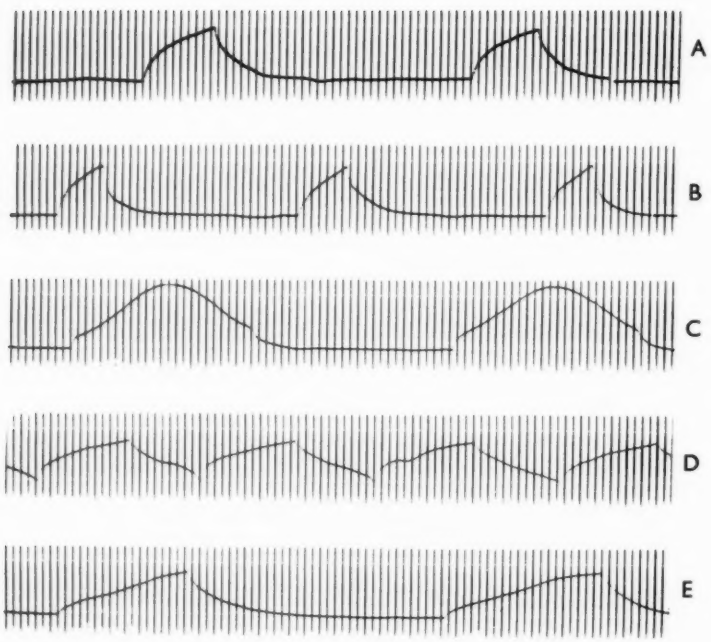

FIG. 4

Time/bronchial pressure tracings using a capacitance manometer and a recording oscilloscope. All from same patient. Tidal volumes between 350–480 ml. Peak pressures between 11–16 cm H_2O. Chest open.

Ordinate = pressure; Abscissa=time (1/10 sec intervals).

A. Barnet respirator (time cycled)

B. Pask respirator (pressure cycled)

C. Beaver respirator with solenoid valves (volume cycled)

D. The Stephenson minute-master (pressure cycled)

E. Pneumotron (time cycled)

Ministry of Health report—Anaesthetic Explosions 1956, Section V, para. 18.

The solenoids and their voltage rectifiers are individually hermetically sealed and the machine when efficiently earthed can safely be used with explosive anaesthetics.

The various units are mounted in an enclosed metal case with the controls upon the front panel. A pointer connected to the moving top plate of the concertina bag is visible through a slot in the panel and its position and range of movement is thus rendered visible. A compartment in the back of the case contains the electrical supply cable and the plastic tubing and connections during transportation (figs. 5, 5A, 5B).

(f) *Power supply.*

The machine can be operated from the mains or from an accumulator requiring 33 watts for piston valves and 6 watts for poppet valves. The use of the latter permits a dry battery to be used for limited periods.

SUMMARY

A time cycled intermittent positive pressure respirator has been described having the following characteristics:

(*a*) Readily portable and operable from either mains or accumulator.

(*b*) Being inserted between an anaesthetic machine or other gas supply and the patient, it will operate from the normal rate and pressure of gas flow used for manual control of respiration by intermittent positive pressure (i.e. approximately 8 1./min at 5 lb./sq.in pressure.

(*c*) Will permit infinitely variable adjustment of the inspiratory/expiratory ratios between 9:1 and 1:9, respiratory rates between 12 and 60 respirations per minute at an inspiratory period 0.5 sec and peak bronchial pressures between 5 cm and 30 cm of water pressure.

(*d*) Will produce bronchial pressure curves corresponding to the natural tidal volume curve and independent control of the length of inspiratory and expiratory periods.

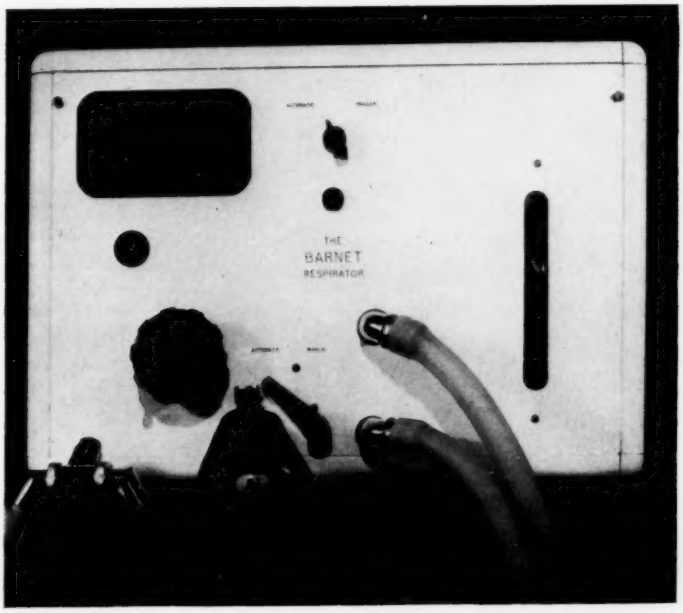

FIG. 5
Exterior view of respirator.

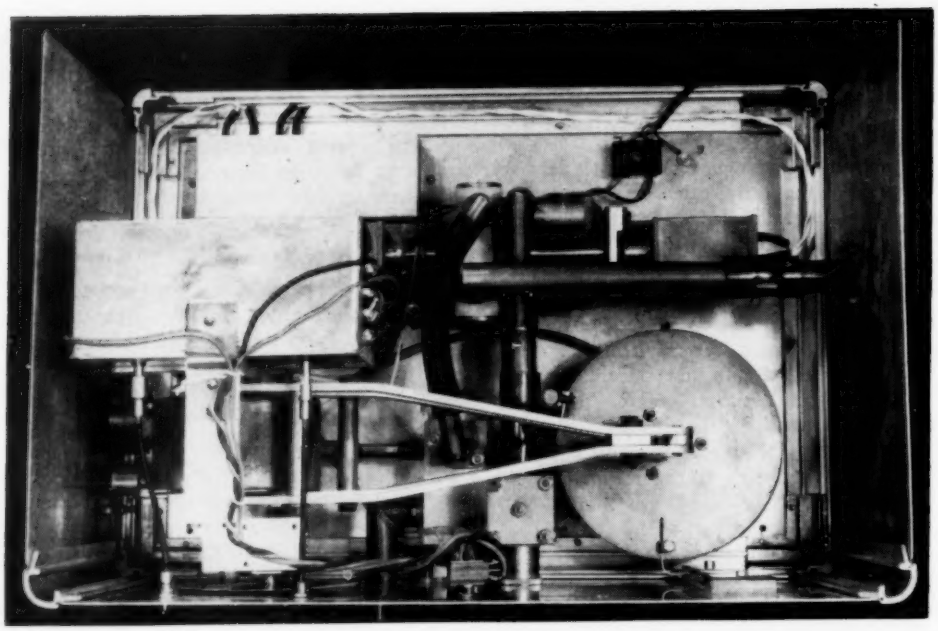

FIG. 5A

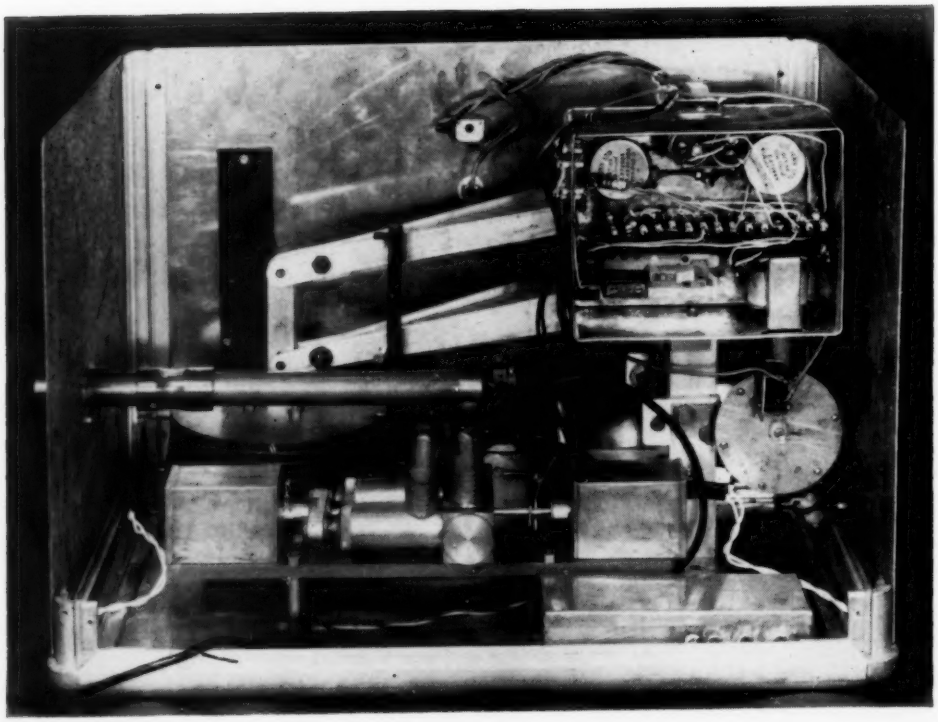

FIG. 5B
Interior views of respirator.

(*e*) A high instantaneous flow rate (80 l./min), permitting a short inspiratory period with adequate ventilation.

(*f*) Operates as a one-way gas circuit without rebreathing.

(*g*) Allows the patient to trigger the machine which then reverts to its own rhythm until triggered again, the filling of the reservoir bag being adjusted to at least twice the tidal volume of the patient.

(*h*) Gives visual warning of respiratory obstruction, the bag slowly or rapidly filling to its maximum capacity with diminished tidal excursions.

(*i*) Permits rapid transfer to manual control of respiration without disconnecting the respirator from the source of gas or the patient.

(*j*) The electronic and electrical circuits being either hermetically sealed or essentially safe circuits, permit the use of explosive anaesthetics or the operation of the machine in a potentially explosive atmosphere.

ACKNOWLEDGMENTS

We wish to thank the Barnet Group Hospital Management Committee for a grant; Messrs. W. Watson and Son Limited for constructing a set of valves; Messrs. British Oxygen Co., Dr. D. M. H. Cogman and Messrs. Pye Radio Ltd. for providing the photographs; and the surgeons of the Barnet Group of Hospitals for their co-operation.

REFERENCES

Cournand, A. (1948). Physiological studies of the effects of intermittent positive pressure breathing and cardiac output in man. *Amer. J. Physiol.*, **152**, 162.

Gaensler, A. E. (1956). Discussion following paper (by Nealon T. F., Heupt, J. G.): Inefficient carbon dioxide absorption requiring increased pulmonary ventilation during operations. *J. thorac. Surg.*, **32**, 464.

Gordon, A. S., Frye, C. W., and Langston, H. T. (1956). The cardiorespiratory dynamics of controlled respiration in the open and closed chest *J. thorac. Surg.*, **32**, 431.

Maloney, J. V. (1954). Problems in ventilation: a panel discussion. *Anesthesiology*, **15**, 416.

Motley, H. L., Cournand, A., Werko, L., Dresdale, D. T., Himmelstein, A., and Richards, D. W. jnr. (1948). Intermittent positive pressure breathing (a means of administering artificial respiration in man). *J. Amer. med. Ass.*, **137**, 370.

Proctor, D. F. (1955). Studies of respiratory air flow. IV: Resistance to airflow through anaesthesia apparatus. *Bull Johns Hopk. Hosp.*, **96**, 49.

Radford, E. P. (1955). Ventilation standards for use in artificial respiration. *J. appl. Physiol.*, **7**, 451.

Spalding, J. M. K. (1955). Pressure and duration of inspiration during artificial respiration by intermittent positive pressure. *Lancet*, **1**, 1099.

BOOK REVIEW

Die Postoperativen Frühkomplikationen: Ihre Behandlung und Verhutung. By Doz. Dr. Kurtwiemers and Dr. Ernst Kern. Published by Georg Thieme Verborg, Stuttgart. Pp. 262. Price D.M.38.

The great reduction in surgical mortality which has taken place since the war has been due mainly to an increasing knowledge of the correct preparation and aftercare of the patient rather than to an improvement in technical skill. The steady spate of articles and monographs has made it difficult for surgeons and anaesthetists to keep up to date with this knowledge and at the same time not to be confused by many of the differing opinions. There is, therefore, a need for a book which can present a balanced picture of modern pre-operative and postoperative care. The subject of this review, the text of which is published in German, attempts to do this.

The authors have managed to cover the whole subject within 250 pages and yet very few subjects are inadequately dealt with. The disturbances of water and electrolyte metabolism and their correction form the major part of the book and are lucidly explained, although in the section on burns Wallace's "Rule of Nine" is made unnecessarily complicated. The care of neurosurgical and thoracic patients is well discussed and the illustrations in these sections are excellent. The section on infections is not very clear and no mention is made of any antibiotic more recent than the tetracyclines. In a book of this length, it would seem inappropriate to spend five pages on tetanus—surely a reflection on Continental asepsis.

On the whole, this book is an excellent comprehensive review of its subject. In addition to the language barrier, it has one major defect in that most of the drugs mentioned are given proprietary names and their true nature and British and American counterparts can only be guessed at.

Anthony R. Anscombe

Brit. J. Anaesth. (1958), **30**, 32

A VOLUME CONTROLLED PATIENT-CYCLED RESPIRATOR FOR ADULTS

With air or anaesthetic gas mixtures

BY

J. R. GREER AND I. DONALD

From the Regional Department of Physics and University Department of Midwifery, Glasgow

THE arguments in favour of augmenting spontaneous but inadequate respiratory effort in the newborn have been discussed elsewhere (*Brit. J. Anaesth.* (1957), **29**, 553). It was natural, as a corollary, that we should extend our enquiry to adults. The desirability of employing a respirator capable of acting synchronously in step with an adult patient's own breathing (if any) has yet to be assessed. At the same time such a respirator should, as in the case of the newborn, be capable of operating automatically at a suitable and predetermined rate if spontaneous respiration fails or becomes dangerously shallow or infrequent.

The apparatus should work preferentially in step with the patient's breathing, and only when the absence of an adequate inspiratory stimulus exceeds a given period, for example 5 seconds, should the artificial timer operate. As soon as spontaneous breathing is resumed the machine reverts automatically to the patient-triggering system.

Such a machine is here described. It follows the same principles as our respirator for the newborn; however, a more satisfactory control of ventilation was obtained in adults by setting the stroke volume of each breath to a predetermined amount. This advantage is less certain with babies in the presence of atelectasis of varying degrees and where the vital capacity cannot be predicted.

So far clinical trials have been limited to patients undergoing gynaecological operations in whom muscle relaxants have been used and, under these circumstances, the steady rhythm of artificial respiration, maintained by the automatic timer, alters as the effects of the relaxant wear off and the return of spontaneous breathing resumes control of the machine.

GENERAL DESIGN

It was decided to copy, as far as possible, the action of the standard manually operated bag and for this purpose a concertina-like bag was mounted inside a perspex pressure casing (figs. 1 and 2). The bottom of the bag is weighted so that it rests firmly on an adjustable platform when the bag is fully opened. The position of this platform thus sets the stroke volume. Delivery from bag to patient is achieved by filling the perspex casing with compressed air and when the pressure of this is released—or a negative pressure applied—the bag refills itself with gas mixture or air as required. If the patient inhales spontaneously, a change in pressure operates a trigger (fig. 3) causing compressed air to be applied to the bag. If, on the other hand, no breath operates the trigger during a preset period, a delay timer comes into action and causes the respirator to operate in the same way.

The compressed air is obtained from a small pump and reservoir. Regulation of the flow of compressed air to the respirator is effected by a needle valve, since the pressure from the pump is kept uniform at about 10 lb. per square inch. The needle valve, therefore, controls the duration of the inspiratory period. As this period can be reduced to under 1 second, if required, the peak flow rate exceeds 50 litres per minute.

Safety Valve.

If the respirator is set to deliver a large stroke volume in a short inspiration time, the pressure required to achieve this might exceed what is clinically desirable. Mushin and Rendell-Baker (1953) refer to pressures of 25 cm H_2O or more as being necessary for efficient ventilation, and suggest an upper limit of 30 cm H_2O. Behnke

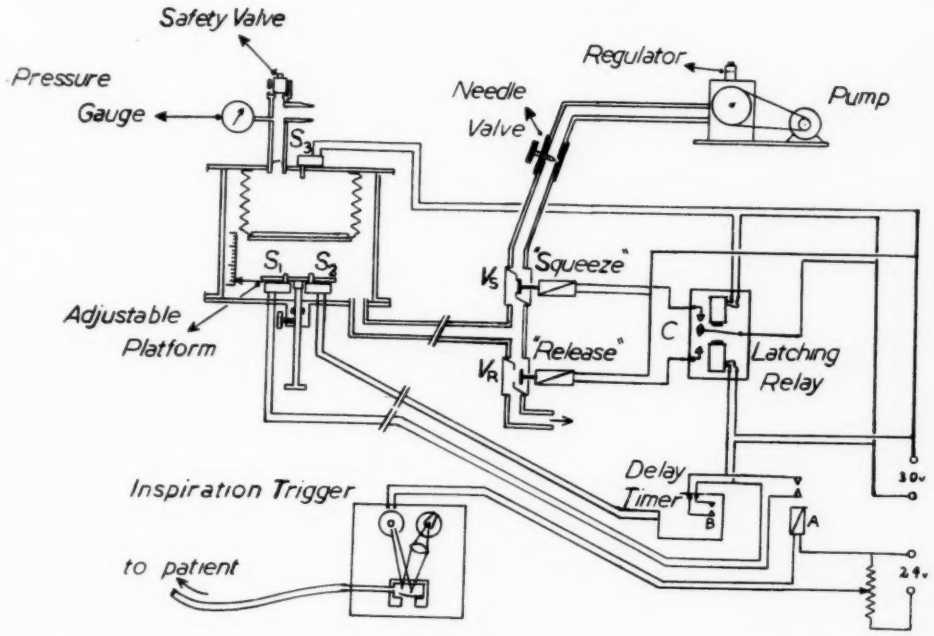

FIG. 1

Schematic drawing of respirator showing (top left) perspex casing with corrugated bag, adjustable platform, and outlet port with safety valve and pressure gauge. In the interests of clarity the inlet port and manifold have been omitted (see figs. 4A, 4B). S_1, S_2, S_3, are Burgess Microswitches; V_S and V_R solenoid valves; A is a P.O. relay type 3000; B a B.T.H. Electronic Timer type FU 21; and C a resettable overload relay (government surplus).

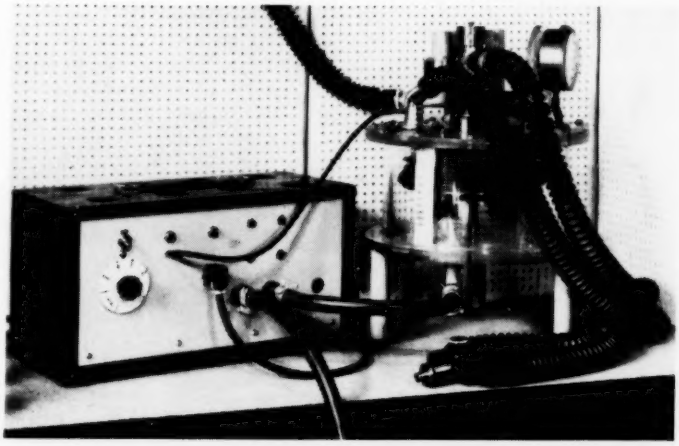

FIG. 2

A volume-controlled patient-cycled respirator for adults.

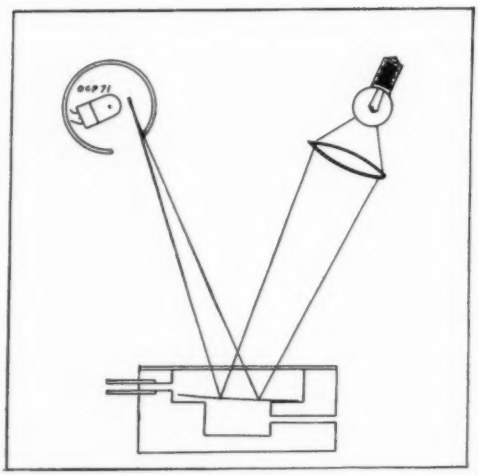

FIG. 3

Detail of photo-electric inspiration trigger, showing aluminized terylene flap partly raised.

(1941) in experiments on relaxed conscious adults reported that intermittent pressures of 15 mm Hg (20 cm H_2O) were acceptable, but 25 mm Hg (34 cm H_2O) produced a sensation of substernal distress. Saklad (1953), quoting this, adds that the pressure required to overdistend the lungs and thus rupture the alveoli of healthy adults would appear to exceed 30 mm Hg (40 cm H_2O). It would therefore seem that an upper limit of safe pressure might reasonably be fixed anywhere between 25 cm H_2O and 35 cm H_2O, but in the present respirator the weight-operated safety valve has been designed to limit pressures to 25 cm H_2O, although this can readily be increased.

Photo-electric Trigger.

The photo-electric trigger used follows closely the design developed for infant respirators. It consists (fig. 3) of a closed perspex cell with a tube leading off one side and a hole in the base covered with a flap of aluminized terylene. Light from a small bulb is focused by a condenser, and reflected by the aluminized terylene on to a phototransistor (type OCP71). While thus illumined the phototransistor passes enough current to hold open the contacts of A, a Post Office relay (fig. 1). When, however, any air is drawn up through the trigger at the beginning of a spontaneous inspiratory effort the flap is lifted, the light deflected, and these relay contacts close. The speed of this action is limited only by the operating time of the relays and solenoid valve (less than 1/10th second), and there is no perceptible delay between the patient's effort and the commencement of the resulting inspiration cycle.

Delay Timer and Latching Relay.

A standard industrial timer controls relay B so that its contacts close at any preset interval up to 5 seconds after switch S2 has been operated by the bag coming to rest on the platform at the end of expiration. The latching relay (C) is stable in either of two positions, following a momentary pulse in either coil. It is used to switch the solenoid valves Vs and V_R and, as a matter of convenience, its coils have been labelled "Squeeze" and "Release" (fig. 1) to indicate their functions in relation to the rubber bag. During the inspiratory phase the bottom of the bag rises off the platform and in doing so opens switch S1, thereby rendering the trigger inoperative until the cycle is completed.

OPERATING CYCLE

The weighted bottom of the bag rests on an adjustable platform, (fig. 1). While in this position it closes switches S1 and S2, simultaneously connecting the photo-electric trigger and initiating the delay timer. If now either relay A or B closes, due to the action of the trigger or the timer, whichever is the sooner, the latching relay swings to "Squeeze", and compressed air is admitted to the perspex casing via the solenoid valve Vs. This squeezes the bag and continues to do so until all the air or gas it contained has been conveyed to the patient (or released via the safety valve). By now the base of the bag presses against the top of the perspex casing, and a third contact (S3) in thus closed. This swings the latching relay C to the release position, and the solenoid valve Vs is shut, cutting off the compressed air, while at the same time V_R is opened, allowing the air in the perspex casing to escape. The weighted base of the bag now falls until it again rests on the platform, so recharging the bag (fig. 4) and at the same time

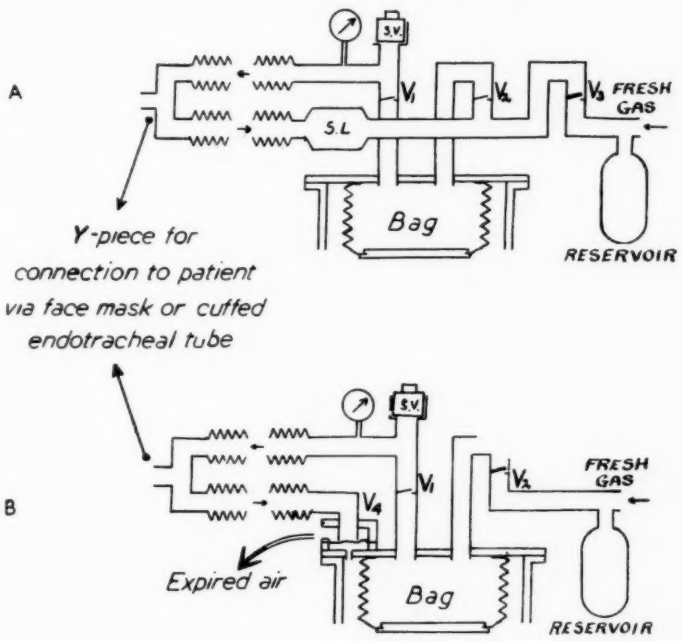

FIG. 4

(A) Connection of respirator to patient for closed circuit operation showing fresh gas inlet on the right, and corrugated tubing to facepiece on the left.

(B) Connection of respirator to patient for total loss operation, showing fresh gas inlet on the right, and corrugated tubing to facepiece and expiratory valve V4 on the left.

preparing for the next cycle by closing S1 and S2.

CLOSED CIRCUIT OPERATION

The apparatus can be arranged to operate in either a " total loss " or a closed circuit system. It was originally tested on the latter and this application will therefore be described first.

Considering the cycle from the point where compressed air starts to squeeze the bag, the gas passes out to the patient through one-way valve V1 (fig. 4A), the pressure being indicated by a pressure gauge. (This is limited to a maximum of 25 cm H_2O by the weight-operated safety valve.)

When the bag is released it is filled by expired air from the patient, after this has passed through a soda lime canister (S.L.). Lost gas, and CO_2 absorbed by the soda lime is replaced by fresh gas or air at atmospheric pressure drawn in through valve V3. The flap of V3 is designed so that a pressure gradient of 5 cm H_2O is required to open it; thus a negative pressure of 5 cm is maintained throughout expiration. A further purpose in raising the operating threshold of V3 is to prevent a gradual build-up of gas pressure within the respirator bag due to the possible leakage of gas accumulating inadvertently under pressure within the reservoir.

The primary use of a closed circuit system is with anaesthetic gases, and it was found desirable to use the respirator in the same manner as a manually operated bag—namely to leave the valve on the Y-piece partly open, and to

work with rather larger stroke volumes than are required by the patient—thus ensuring a continual replenishment of the gas mixture, and preventing a progressive alteration in the proportion of the component gases, as a result, for example, of oxygen uptake.

"TOTAL LOSS" OPERATION

Partly because of the difficulty of estimating the precise nitrous oxide/oxygen mixture in a closed circuit, and partly in view of reports of a lower incidence of postoperative hangover among patients where a total loss technique was employed, it was decided to modify the apparatus to use this method as an alternative. Apart from its advantages with trichloroethylene, such a system is, in any case, clearly preferable to a closed circuit when atmospheric air alone is being used.

The main alteration concerns the return tube from the patient to the bag. In the closed circuit (fig. 4A) pressure from the bag held the non-return valve (V2) closed during the inspiratory phase, ensuring that all the gas reached the patient; while, during expiration, the same valve permitted expired air to pass from the patient back to the bag. This valve was therefore replaced by another (V4), with a corresponding function (fig. 4B). This valve consists of a thin rubber membrane lightly applied over the return tube from the patient. The membrane is connected with the perspex pressure casing. During the inspiratory phase, compressed air is admitted to the perspex casing, squeezing the bag and, simultaneously, pressing the membrane firmly against the end of the return tube, thus preventing loss of gas. At the end of inspiration, the compressed air is shut off, the membrane relaxes, and the patient is free to exhale to atmosphere.

As there is now no return path from the patient to the bag, it is no longer possible to obtain the negative phase from the falling bag, and it would instead be necessary to derive this from the low pressure side of the pump—in some respects a less elegant solution. On the other hand, the absence of an intermittent negative pressure in the return tube makes it possible to connect the trigger directly to the return tube from the patient with a substantial increase in sensitivity.

SUMMARY

A respirator is described which, it is believed, represents a new approach to the problem of ensuring optimal ventilation of certain difficult cases. This is achieved by a dual timer-trigger system, coupled with a variable rate of delivery, which may be increased to over 50 litres/minute. Two forms of the respirator are considered, one for use in a closed circuit, the other for "total loss" operation: each has been used successfully throughout surgical operations in which relaxants were used, the return of normal breathing coinciding with a smooth change-over from automatic to patient-triggered operation. The apparatus has not as yet been sealed against explosive gases.

ACKNOWLEDGMENTS

We are indebted to Dr. J. Crawford and Dr. H. Wishart, anaesthetists at the Western Infirmary, Glasgow, for advice, and for their close co-operation during clinical tests; also to Mr. J. Macfadyen of the Regional Physics Department, Glasgow (where the apparatus was built), who assisted in the design and carried out all the constructional work. The development is a by-product of a research programme on neonatal respiratory disorders supported by a grant from the Scottish Hospital Endowments Research Trust, to whom grateful acknowledgments are made.

REFERENCES

Behnke, A. R. (1941). Certain physiological principles underlying resuscitation and oxygen therapy. *Anesthesiology*, **2**, 245.

Mushin, W. W., and Rendell-Baker, L. (1953). *Principles of Thoracic Anaesthesia*, p. 15. Oxford: Blackwell Scientific Publications.

Saklad, M. (1953). *Inhalation Therapy and Resuscitation*, p. 248. Oxford: Blackwell Scientific Publications.

Brit. J. Anaesth. (1958), **30**, 37

VOLATILE ANAESTHETICS

A Method for their Controlled Administration

BY

E. FALKNER HILL

WHEN Johnstone (1956) anaesthetized his first 500 cases with halothane, he passed a mixture of oxygen and nitrous oxide over the halothane in an ordinary trichloroethylene bottle with plunger at the top. Subsequent examination of this bottle showed that he had been using 0.5–2.5 per cent according to the position of the lever.

Mapleson (1957), using a similar bottle with trichloroethylene, found that considerable variation in the percentage of vapour coming over due to play in the plunger bearing. Hillard (1957) suggested a modification which remedied this. The British Oxygen Co. substituted a single tube with blocked end but side opening about 1 cm above the surface of the liquid. When 2 litres of oxygen per minute were passed over halothane in such bottles the results shown in table I were obtained.

TABLE I

Time	Temperature °C	Percentage	Percentages given by bottle 2
9.51	21.0	7.3	6.7
9.54	18.0	6.1	5.95
9.58	16.0	5.4	—
10.02	14.5	4.8	5.0
10.06	13.0	4.5	—
10.10	12.5	4.2	—
10.14	11.5	4.1	4.25
10.18	11.0	4.0	—

Both bottles were of the same type and if the average of a number of experiments is taken the figures approximate more closely than those given in the table above. If now 2 litres are passed round the bottle containing the halothane to mix with the 2 litres passed over the halothane I expected that the percentages would be halved, but in neither case was the expectation realized. A typical result is shown in table II.

TABLE II

Temperature, °C	Percentage	Expectation	Realization
21.0	7.3	3.6	2.5
18.0	6.1	3.0	2.0
16.0	5.4	2.7	1.7
14.5	4.8	2.25	1.6
13.0	4.5	2.25	1.5

To anyone entering the laboratory the odour of halothane was noticeable and I had no doubt that the bottles were leaking. Many years ago, when experimenting with $CHCl_3$, I had had the same difficulty with leaks. As $CHCl_3$ is less volatile and less costly than halothane I started a number of experiments with $CHCl_3$. Messrs. Cowlishaw and Co., expert scientific glass manufacturers, made a bottle for me of about 300 ml capacity, with fused-in entry and exit tubes, and a central ground glass stopper holding a thermometer which extended down into the liquid. There is here, then, no possibility of a leak. This time expectation and realization are indistinguishable (table III).

TABLE III

Temp. °C	$CHCl_3$ with 2 l. oxygen passing over it %	$CHCl_3$ with 2 l. over and 2 l. round %
21.0	5.1	2.5
17.5	4.5	2.1
13.5	3.9	2.0
13.5	3.6	1.8
12.5	3.5	1.75
11.0	3.3	1.66
10.5	3.29	1.58
9.5	3.0	1.5

Similar figures were obtained when 2 litres over were mixed with 3 litres round, giving two-fifths of the undiluted vapour and again when 2 litres over the $CHCl_3$ were diluted with 1 litre

round, giving two-thirds of the undiluted vapour. The figures are shown in table IV

TABLE IV

Temp. °C	Percentages		
	Undiluted	Calculated	Found
$CHCl_3$—2 l. over, 3 l. round (2/5ths)			
21.0	5.1	2.0	2.1
18.0	4.4	1.7	1.6
16.0	3.8	1.5	1.6
13.5	3.4	1.36	1.4
12.5	3.2	1.28	1.3
2 l. over, 1 l. round (2/3rds)			
21.0	5.1	3.4	3.25
18.0	4.4	2.9	2.8
16.0	3.8	2.5	2.6
13.5	3.4	2.3	2.4

These results were sufficiently satisfactory to make it worth while to use the same bottle for the estimation of halothane percentages coming over with the passing of oxygen at similar rates and proportions over the halothane and round it. Some of the figures obtained are given in tables V and VI.

These figures, together with the clinical experience at our disposal, would seem to suggest that halothane is a somewhat more powerful anaesthetic than $CHCl_3$ and the concentrated vapour should be diluted with three times its volume of oxygen. In practice the following arrangement is desirable, as the nicest modifications in the percentages coming over are instantly made. A source of oxygen (the usual cylinder) communicates with a large vessel, a Winchester quart or, better still, a 2 litre bag. This leads to a Y shaped glass tube with taps on each arm of the Y. Each arm leads to a flowmeter, and from thence to the patient. One flowmeter passes its gas, via the bottle containing the halothane, to one arm of a second Y tube, whilst the other passes its oxygen to the other arm of this second Y. They mix in the stem and so reach the patient. The first thing to decide is the flow the anaesthetist wishes to use, say 4 litres a minute. All these litres go to the flowmeter taking the oxygen round the bottle. The tap on the arm of the Y communicating with the flowmeter carrying the oxygen going to the bottle containing the halothane is then turned on till it registers 1 litre per minute. The other flowmeter will be seen to be registering only 3 litres per minute. The patient will be receiving 1.7 to 1.2 per cent according to the temperature. By turning the tap on the "free" flowmeter so that 2 litres per minute are flowing through each of the flowmeters the percentage going to the patient will be increased from 3.5 to 2.5 per cent again according to the temperature (table VII). It takes a quarter of an hour to describe: it is done simply by turning a tap.

By diluting the quantities shown in table VIII with oxygen passing round the bottle containing

TABLE V

Halothane 1 litre per minute over halothane.

Time	Temp. °C	%	With 2 l. round		With 3 l. round	
			Calc. %	Found %	Calc. %	Found %
10.15	21	6.5				
10.19	19	5.6	1.9	2.2	1.4	1.5
10.24	18	5.0	1.7	2.0	1.26	1.3
10.28	17	4.8	1.6	1.7	1.2	1.2
10.32	17	4.8				
10.37	17	4.7				

TABLE VI

Halothane 2 litres per minute over halothane.

Time	Temp. °C	%	With 4 l. round	
			Calc. %	Found %
11.51	21.0	6.9	2.3	2.5
11.55	17.5	6.2	2.1	2.2
11.59	15.5	5.8	1.9	2.0
12.3	14.0	5.4	1.8	1.9
12.7	13.5	5.1	1.7	1.8

TABLE VII
2 l. over and 2 l. round.

Temp. °C	Percentages	
	Calculated	Found
21.0	3.45	3.5
17.5	3.0	3.0
15.5	2.8	2.7
13.5	2.6	2.5
13.5	2.5	2.5

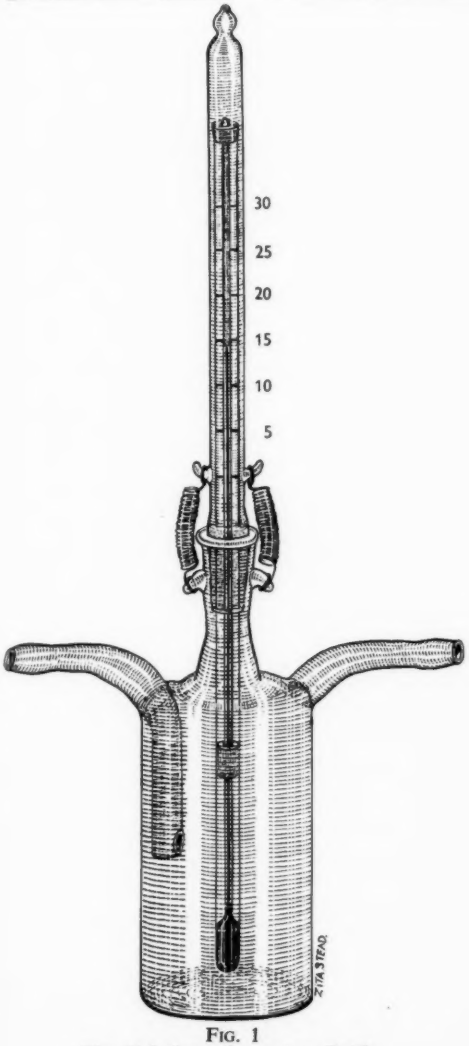

FIG. 1
The Halothane Percentage Bottle.

the halothane the anaesthetist can get any percentage he requires merely by turning the tap regulating the flow of the diluting oxygen.

TABLE VIII
Oxygen at various rates of flow over halothane.

Rate per min.	Percentages at		
	21°C	17°C	10°C
2 litres	7	–	5
1 litre	6	4	–
500 ml	5	4	–

Here are appended three typical experiments (table IX). They give an idea of the accuracy of the method and are the result of close on three hundred observations.

TABLE IX

Temp. °C	Undiluted	Calculated	Found
Halothane—2 litres over, 4 litres round (1/3)			
21.0	6.9	2.3	2.5
18.0	6.2	2.1	2.2
15.5	5.8	1.9	2.0
14.0	5.4	1.8	1.9
13.5	5.1	1.7	1.8
1 litre over and 2 litres round gave the same figures as above.			
Halothane—1 litre over, 3 litres round (1/4).			
21.0	6.5	1.6	1.7
19.0	5.6	1.4	1.5
18.0	5.0	1.25	1.3
17.0	4.8	1.2	1.2

The bottle, flowmeters, taps and connections have been enclosed in a box which renders the whole apparatus portable. It weighs about 10 lb. (4.5 kg) and needs only an oxygen cylinder with reducing valve at one end and a patient at the other to get into action. The inlet and exit tubes are supplied with taps.

The makers are Messrs. Cowlishaw Ltd., 42 Bridgeford Street, Manchester 15.

REFERENCES

Johnstone, M. (1956). The human cardiovascular response to Fluothane anaesthesia. *Brit. J. Anaesth.*, **28**, 392.

Mapleson, W. W. (1957). Trichloroethylene concentration from a "Boyle" type anaesthetic apparatus. *Brit. J. Anaesth.*, **29**, 3.

Hillard, E. K. (1957). A modification to the trichloroethylene vaporizer on the "Boyle" apparatus. *Brit. J. Anaesth.*, **29**, 39.

ON CHLOROFORM AND OTHER ANÆSTHETICS

THEIR ACTION AND ADMINISTRATION

BY

JOHN SNOW, M.D.
Licentiate of the Royal College of Physicians

(*Continued from Vol. XXIX, page* 526)

Dr. Fenwick found that the mortality from lithotomy had been greater since the introduction of chloroform than before, but that the increase was confined to the cases occurring in adults; and he justly attributes this to the fact of the more favourable cases having been operated on by lithotrity. He says, "However paradoxical it may appear, I believe that as surgery improves the general average of mortality, both after amputations and lithotomy will increase; in the former from the operation being confined more and more to those suffering from accidents, and in the latter, from those with a healthy condition of the kidneys and bladder being selected for the action of the lithotrite, and the worst cases only submitting to the knife."

Dr. Fenwick gives a table showing the causes of death in the fatal cases of lithotomy before the use of chloroform and afterwards, by which it is seen that, in the latter set of cases a larger proportion of the patients had been afflicted with organic disease, in addition to the stone. He says:—"This table shows that the relative proportion of deaths arising from the operation in healthy persons has diminished since the use of chloroform, 58 per cent. having died in the former and only 47 per cent. in the latter."

Dr. Fenwick inquires into the result of a considerable number of operations in the Newcastle Infirmary, before and after the use of chloroform, in addition to those mentioned above; and gives the following summary towards the end of his paper.* "We find that there has been a decrease in mortality since the introduction of chloroform in the following operations:—

	Per cent.
Pathological amputations of the thigh ...	5
Traumatic amputations of the thigh... ...	17
Pathological amputations of the leg ...	8
Amputation at the shoulder joint	7
Traumatic amputation of the arm	5
Pathological amputation of the forearm ...	33
Traumatic amputation of the forearm ...	16
Ligature of the brachial artery	20
Ligature of the arteries of the forearm ...	25
Amputation of the penis	11
Amputation of the testis	14
Excision of the elbow	30
Tumours of the axilla, etc.	8
Tumours of the bones, etc.	4

"There has been an increase of mortality in:—

	Per cent.
Traumatic amputation of the leg	5
Pathological amputation of the arm ...	41
Lithotomy	10
Herniotomy	12
Amputation of the breast	1
Tumours of the head, etc	5

Dr. Fenwick very properly remarks that other circumstances may have had an influence over the mortality of the various operations, as well as the fact of chloroform being used or not used: but his inquiry is very important, as it shows the

* Page 648.

result of operations in the same institution since the employment of ether and chloroform, and for a long period immediately preceding the use of those agents.

THE ADMINISTRATION OF CHLOROFORM IN THE DIFFERENT KINDS OF OPERATIONS

General directions for the exhibition of chloroform have already been given, but it is desirable to say a few words regarding its employment in some of the individual operations of surgery.

Lithotomy. In this operation, it is advisable to have the patient placed on the operating table with his head supported by one or two pillows, and to administer the chloroform before the sound is introduced. This having been done, it is generally requisite to administer a little more vapour cautiously and gently, whilst the bandages are applied to the hands and feet, and the patient is drawn down towards the foot of the table. The bandaging should on no account be neglected in the adult patient. It would be an abuse of chloroform to carry its effects so far in every case that the slightest contraction of the muscles could not be excited by the use of the knife, merely to save the trouble of applying a bandage. In little children, the bandages are not usually employed; but in these cases, the person administering the chloroform should hold the head or shoulders of the little patient, just as the surgeon makes his first incision; for the two assistants who are holding the legs, and think they are doing a great deal of good, are really making a fulcrum to enable the child to push itself away from the surgeon, if it makes the least flinch, which it is almost sure to do, if the narcotism is not much deeper than there is any occasion for. In this operation the patient should be fully insensible when the surgeon makes his first incisions; and the chloroform must be repeated or not during the operation as occasion may require.

I have memoranda of fifty-seven cases in which I have administered chloroform in the adult, and thirty-four in children, down to the present time, the end of March 1858. It was my expectation that I should be able to give the result of these, and all the other important operations; but I have in a few cases either not ascertained whether the patient recovered or not, or have neglected to make a note of the circumstances at the time; and although the deficiencies might perhaps be made up by private correspondence, and by searching the books of three hospitals, I regret that I have not time at present for such an undertaking.

A large proportion of the above cases of lithotomy were performed by Mr. Fergusson, either in his private practice, or in King's College Hospital, and the remaining operations were performed by a number of surgeons. On April 19th, 1855, Mr. Fergusson removed eight calculi, as large as pigeon eggs, from the bladder of a gentleman, aged sixty-nine, who had a favourable recovery.

On June 7th, in the same year, he removed fourteen calculi from the bladder of an elderly gentleman, who also recovered. In January 1857, he removed forty-two calculi from the bladder of a gentleman, aged sixty-six; twelve of them were nearly as large as chestnuts, and the rest about as big as hazel-nuts. This patient died, I think, within a fortnight.

He had disease of the kidneys. A considerable number of the elderly men on whom lithotomy has been performed have had well marked symptoms of fatty degeneration of the heart; but the chloroform has acted favourably on all of them. In January 1853, Mr. Fergusson performed lithotomy in the country on a gentleman, aged sixty-four, removing a rather large calculus. The patient weighed eighteen stone; he had a pulse which was liable to intermit, some of his relations had died suddenly, and his usual medical attendant was very much alarmed about the chloroform; but its action was very satisfactory. The patient recovered his consciousness without sickness, or any unpleasant symptom; and he had a favourable recovery. On August 11th, 1855, I administered chloroform in King's College Hospital to a man, aged seventy-five, whilst Mr. Fergusson performed lithotomy. The patient had suffered from apoplexy a year before; he was still paralyzed on one side, he had the arcus senilis well marked in both eyes, and the temporal arteries were very thick and rigid. He went through the operation well; the pulse being good all the time. He died on the fifth day, and the following appears in the *Medical Times and Gazette* respecting the appearances found after death. "At the autopsy, the cellular tissue of the pelvis was found infiltrated with pus. The kidneys were contracted, and occupied by many cysts; the ureters were inflamed and contained pus. The bladder was extensively sacculated, and its mucous membrane so much congested as to appear on the point of sloughing. There was no peritonitis. The heart was large and fatty, and its aortic valves diseased to a small extent. The brain was congested, and its blood-vessels extensively ossified." On December 22nd, 1855, I administered chloroform in King's College Hospital to a very fat man, aged sixty-one, with a soft weak pulse, and arcus senilis of the cornea. Mr. Fergusson performed lithotomy. He went well through the operation, and recovered promptly from the effects of the chloroform. He died on the seventh day, with purulent effusion into the left knee, and around the left shoulder. The heart was large and flabby, and the substance of the left ventricle was very friable. The right ventricle was dilated and thinned, and its walls were soft and friable, and encroached on externally by fat; at one place there seemed scarcely any muscular substance left.

I have memoranda of four cases of lithotomy in the female, in which the urethra was divided as well as dilated to extract the stone; and I recollect two other cases of the same kind of which I have no notes.

Lithotrity. I have memoranda of 155 operations of lithotrity in which I have administered chloroform. The number of cases of stone in which these operations took place was sixty, but some of the patients had operations performed, either without chloroform, or when it was administered by some one else. The earliest cases of lithotrity in which I administered chloroform were in St. George's Hospital, and a great number of the patients to whom I have given it were the private patients of Mr. Fergusson, who is entirely satisfied of its utility in this operation. Speaking of this operation in his *Practical Surgery* (third ed., p. 800), he says: "I am of opinion that there is not

any department of practical surgery in which anæsthesia has been of more service than in this." I have administered chloroform in a few operations of lithotrity by Sir Benjamin Brodie; but I believe that he does not approve of it in this operation, as a general rule. The following are amongst the other surgeons whom I have frequently assisted by giving chloroform in this operation: Mr. Cæsar Hawkins, Mr. Cutler, Mr. Coulson, Mr. Charles Hawkins, and Mr. Henry Lee. Mr. Fergusson, in one of his operations, extracted some portions of gutta percha bougie round which a phosphatic calculus had formed. He was not informed of the presence of the bougie in the bladder, until it was extracted. On another occasion, a patient was brought from the country soon after a bougie of this description had broken off, and he extracted with the lithotrite the piece remaining in the bladder. It was between two and three inches in length.

It is generally desirable to make the patient quite insensible during the operation of lithotrity; and the patient sometimes groans during its performance, when he is in a condition that, so far as one can judge, he would show no sign of sensation under the use of the knife; but he, of course, has no recollection of the operation afterwards. The bladder is sometimes apt to expel the water which is injected, if the patient is not well under the influence of the chloroform; but when the vapour has been continued for a few minutes, and the narcotism is complete, the bladder will, I believe, always hold water as well as if the operation were performed in the waking state, and generally much better. There are some surgeons who occasionally raise the breech of the patient for a short time during this operation, so that the head becomes considerable lower than the body. Stout old men generally snore more loudly when in this position under the influence of chloroform; but I have not met with any ill effects, nor expected any, from the head being lowered for a short time. From ten to twenty minutes has been the most usual duration of the operations under chloroform at which I have been present.

I have administered chloroform six times in female children whilst Mr. Fergusson performed lithotrity. The patients were three in number. I think the calculus all came away after the first operation, and the second was only an examination of the bladder. I administered chloroform also, on one occasion, to a lady whilst Mr. Spencer Wells repeated the operation of lithotrity.

Section of the Urethra in the Perinæum. I have notes of 56 cases of this operation. A great proportion of them were performed by Mr. Fergusson, either in King's College Hospital, or in private practice. Amongst the other surgeons whom I have assisted in this operation were the late Messrs. Guthrie, Bransby Cooper, and Avery. In a great number of the cases, it was impossible to get a catheter into the bladder until after the incision was made in the perinæum, and many of the operations were very tedious; several of them lasting above an hour. It is very desirable to apply the bandages in this operation, as in that of lithotomy, especially if the assistants are not numerous.

Other Operations for Stricture, etc. I have notes of four operations in which the stricture was divided internally by a ure-

thratome; and of two operations in which the orifice of the urethra was enlarged; and of nine plastic operations to restore deficiencies of the walls of the urethra, congenital or otherwise. I have memoranda of 66 cases in which I have administered chloroform for sounding the bladder or the introduction of a catheter. In catheterism under chloroform the patient, of course, lies on his back, which is indeed the position in which he ought to be during this operation, when chloroform is not administered.

Amputation of the Thigh. This operation is often performed for disease of the knee, when the joint is in such a state of tenderness that the least motion causes great pain. In such cases, I have administered the chloroform to the patient in bed, before his removal to the operating table, and given a little more chloroform just before the operation was commenced. In King's College Hospital, I have several times given the chloroform in the ward in such cases, before the patient was removed to the operating theatre; but in St. George's Hospital, the passages and doors being wide, the patient has been carried on his bed into the theatre, where the chloroform was administered just before his being lifted on the table. In those cases in which the tourniquet is applied, in preference to pressure with the fingers, it should be adjusted as the patient is getting under the influence of the chloroform, and tightened just before the operation begins. It is desirable to keep the patient quite insensible till the limb is removed, and the femoral artery is tied; after which a little chloroform may be given whenever the patient shows by a slight flinch, or contraction of his features, that sensation is returning. A few patients have recovered their consciousness during the tying of the smaller arteries, and have entered into a conversation without feeling the pain, but this condition is quite the exception. I have notes of 49 cases of amputation of the thigh in which I have administered chloroform. Some of the patients, who were almost grown up to the adult age, did not know that the limb was removed till three or four days had elapsed.

Amputation of the Leg. I have notes of 31 cases in which I have administered chloroform during this amputation. The remarks made above with regard to amputation of the thigh are applicable to this operation.

Amputation of the Arm. I have only administered chloroform five times in this operation, and only in one case during the last seven years. This case was that of a boy, aged sixteen; Mr. Hancock was the operator, and the patient recovered. I have no note of the disease for which the operation was performed, but in two or three of the remaining cases, the arm was amputated on account of malignant disease of the forearm. In one of the operations, the patient was seated in an easy chair, but in the others, the patients were lying on a sofa, or operating table, with the head and shoulders a little raised.

Amputation of the arm has apparently been much less frequently performed of later years, owing to the increasing practice of performing excision of the elbow; and it appears to me that all the great amputations are much less frequently performed now than they were a few years

ago. During a little more than nine months of 1847, I administered sulphuric ether in 32 amputations of the thigh, leg, and arm; and in the last ten years and four months, I have only administered chloroform in 85 of the same operations; in the last three years, indeed, in only 16 cases; so that the practice of amputation is still diminishing. This is due to the practice of anæsthesia, which enables the surgeon to explore and to remove diseased joints and portions of diseased bone by operations that would be too long and too painful to be endured in the waking state. This circumstance confirms the remark of Dr. Fenwick, previously quoted, that the mortality after amputations may be expected to become greater, as they will cease to be performed, except after accidents, or in very desperate cases.

Amputation of the Ankle. I have notes of 15 cases of amputation at the ankle. They were chiefly performed by Mr. Fergusson, and generally in the manner recommended by Mr. Syme, or nearly so; but latterly Mr. Fergusson and Mr. Partridge have, in three or four cases, adopted the modification of this operation introduced by Prof. Pirogoff, by which a portion of the os calcis is left.

Other Amputations. I have notes of nine cases of amputation of the forearm; four of these operations were performed in 1850, and only three since that year. I have memoranda of twelve cases in which I have given chloroform whilst Chopart's or other partial operations of the foot were performed. Also of three cases in which Mr. Fergusson performed amputation at the knee, and three cases in which he performed amputation at the wrist in King's College Hospital. I have notes also of 65 cases in which I have administered it during the amputation of one or more fingers or toes.

Operations for Necrosis. Anæsthesia is of the utmost service in these operations, which are often tedious, and would be of the most painful nature. The operations are of the most successful kind of any in surgery; they usually relieve the patient from a very painful affection, and leave no mutilation. I have administered chloroform in many cases of necrosis in which an operation could not have been undertaken without its assistance; and in a still larger number, in which the operation could not otherwise have been satisfactorily completed. I have memoranda of 197 cases of necrosis in which I have given chloroform. In 70 of these, the tibia was the bone affected; in 15 cases, the femur; in 24 cases, the humerus; in 14 cases, the radius, or ulna, or both of these bones; in 29 cases, the bones either of the carpus or tarsus, or metacarpus or metatarsus. I have notes of six cases of necrosis of the sternum; in five of these the operation was performed by Mr. Fergusson, and in one case by Mr. Solly. Great care was required in some of the cases to avoid going through into the chest with the bone nippers. There have been nine operations for necrosis of the os calcis. In one of these, Mr. Wm. Adams lately removed, along with the necrosed bone, a small bullet which had been in the bone for six years.

The other cases of necrosis comprise the upper and lower jaw, the parietal bone, the edge of the orbit, the scapula and clavicle, the sacrum, the ileum, and the phalanges of the fingers and toes.

Excision of the Head of the Femur. I have notes of five cases of this operation. They all occurred in children from eight to thirteen years of age. Three of the operations were performed by Mr. Fergusson, one by Mr. Bowman, and one by Mr. French; I also recollect assisting Mr. Henry Smith in a similar operation in a child in 1848, at a time when I did not keep any account of the operations in which I administered chloroform. In one of Mr. Fergusson's operations, he removed some diseased portions of the acetabulum.

Excision of the Elbow. There is every reason to conclude that this excellent operation has been performed more frequently in consequence of the inhalation of narcotic vapours than it otherwise would have been. I have memoranda of 19 cases of this operation in which I have exhibited chloroform; 18 of them were performed by Mr. Fergusson, and the remaining one was performed by Mr. Coulson, whilst Mr. Fergusson was present.

Excision of the Knee. I have notes of 17 cases in which I have given chloroform during the performance of this operation. Thirteen of the operations were performed by Mr. Fergusson, two by Mr. Bowman, one by Mr. Partridge, and one by Mr. Henry Smith.

This operation had been rarely performed till within the last seven years, and there is every reason to believe that it never would have been frequently performed, if the practice of producing anæsthesia had not been introduced. I shall speak of this operation again in treating of amylene.

Excision of Wrist. There have been two cases of this operation performed by Mr. Fergusson in King's College Hospital.

(*To be continued*)

CORRESPONDENCE

A BLOODLESS FIELD WITHOUT HYPOTENSION

Sir,—I write to ask whether any of your readers have encountered a phenomenon which, if it prove regularly obtainable, may be a more acceptable method of achieving a bloodless field of operation than those at present in use. The phenomenon consists of a slight fall in systolic blood pressure (10–20 mm Hg), without alteration of the diastolic pressure, occurring in patients who are horizontal, and associated with a virtually bloodless field.

I have seen this state in patients who have been given ordinary premedication plus chlorpromazine or promazine 30–50 mg, intramuscularly, 1½ hours before operation, and who have received at operation a very dilute and slow intravenous infusion of one of the hypotensive drugs. The technique of anaesthesia appears to be immaterial.

In a recent lumbar sympathectomy, this phenomenon was obtained for 30 minutes with 70 ml of a 0.05 per cent solution (35 mg) of phenacylhomatropinium (Trophenium), the total haemorrhage being less than 10 ml. In another case, one in which the middle ear was being explored and haemorrhage was interfering with the surgery, excellent haemostasis was maintained for 3 hours with 380 ml of a 0.05 per cent solution (190 mg) of camphor sulphonate (Arfonad).

In my few cases, the reversal of the state has been immediate on termination of the infusion, tachyphylaxis has not occurred, and the phenomenon has been seen in young adults with moderate blood pressures, such as are usually resistant to the more orthodox methods of hypotension.

Since there are few cases in my practice in which deliberate hypotension is induced, I should be very glad to know if others have encountered this peculiarity.

Because, in this condition, blood flow in some parts, if not the whole, of the body is obviously altered, there is a potential risk of ischaemic damage to vital organs; however, with a blood pressure altered but little and the patient horizontal, it seems possible that the risk is less than with other techniques of induced hypotension. If others find that the phenomenon can be produced regularly, it may well prove a useful technique in suitable cases.

M. H. Armstrong Davison
Newcastle upon Tyne

COMPARISON OF SEVEN INTRAVENOUS ANAESTHETIC AGENTS IN MAN

Sir,—Dr. J. S. Ruddell has commented in your correspondence column (*Brit. J. Anaesth.* (1957), **29**, 472) on an apparent inconsistency between our conclusions reached in an article published in your Journal and one entitled "The Physiological Effects of Intravenous Anaesthesia on Man" in the *Canadian Anaesthetists' Society Journal.*

May I emphasize first that the paper in the *Canadian Anaesthetists' Society Journal* analyzes a number of additional parameters which were not touched upon in the other publication. I am making this point because from the wording of Dr. Ruddell's letter it might be erroneously concluded that the same experiments were republished in the *Canadian Anaesthetists' Society Journal.* This is not so. As far as tidal volumes and minute volumes of respiration are concerned, hexobarbitone compared favourably with the other agents, and this is brought out in the respective tracings which are reproduced in the publication. However, there was marked irregularity of breathing patterns, crowing, periodic respiration and spontaneous movements of the extremities—all of which were discussed in the first paper. These were incidental manifestations to which we referred in the statement to which Dr. Ruddell objects.

The paper in the *Canadian Anaesthetists' Society Journal* dealt in addition with cardiac output, oxygen consumption, etc., factors which were not being considered in the other publication. Respiration was discussed only from the point of view of adequacy or otherwise of pulmonary ventilation, which indeed was satisfactory.

It appears that the misunderstanding arises from the fact that we were not sufficiently specific

in our statement "especially on respiration". We should have emphasized that this referred to the respiratory pattern rather than the respiratory exchange.

GORDON M. WYANT
Saskatoon, Canada

NITROUS OXIDE BYPASS

Sir,—There are still numbers of anaesthetic machines in use which have a bypass for nitrous oxide as well as one for emergency oxygen. The control levers are identical, and it can easily happen that in the stress of an emergency the wrong bypass is opened when it is intended to inflate a patient's lungs with oxygen. At least one fatality has been caused in this way. I suggest that lives may be saved if every anaesthetist who uses a machine that has a nitrous oxide bypass takes steps to have it removed.

H. R. YOUNGMAN
Cambridge

BOOK REVIEW

The Century of the Surgeon. By Jurgen Thorwald. Published by Thames and Hudson. Price 25*s*.

The author speaks in the first person as though he personally were present at the birth of the new ideas and the practice that flowed from them. In this way he has been able to clothe the bare facts with the warmth of the human feelings and the heat of the human passions that their advent engendered. A very vivid and exciting narrative is the result. He begins with the pre-anaesthetic period when the pain of stone, of intestinal obstruction, of peritonitis, had to be suffered until death put an end to it. The great blessing of anaesthesia, strange to say, was a mixed blessing. It naturally led to an enormous increase in the number of surgical operations and a very high death rate. Septicaemia, pyaemia, gangrene claimed their victims by tens of thousands. In 1847, only one year after Morton had successfully used ether as an anaesthetic, Semmelweis inaugurated the washing of hands in chlorine water as a precaution against puerperal fever. He was appointed Professor of Midwifery in Buda-Pesth in 1855 and the mortality from puerperal fever went down to 0.39 per cent. Meanwhile at Wurzburg, where "the powers that be" would have none of Semmelweis's new-fangled ideas, the mortality from puerperal fever was 26 per cent. Semmelweis recognized puerperal fever as a form of septicaemia, so that not only did tens of thousands of women die in childbirth but a much greater number of men and women died unnecessarily from septicaemia following surgical operations because of the refusal of the majority of the members of the medical profession to see the light.

In 1867 Lister gave his address on "The Antiseptic Principle in the Practice of Surgery" at the meeting of the B.M.A. in Dublin. In spite of opposition and ridicule the idea was gradually accepted and conquered wherever surgery was practised, transforming the hospitals from stinking charnel houses to pleasant places where suffering was mitigated and health restored.

The book has many lessons for us. It shows the terrible price in human lives that has to be paid for certain common human characteristics —the pride, jealousy and stupidity more particularly of "the powers that be". It is a powerful illustration of the oneness of mankind, the brotherhood of man; it shows that the great ones of the earth are obscure people who get on with their work, always opposed, generally ridiculed, more often than not despised, but as a rule only honoured when they are dead. No nation has a monopoly of them; here we have Semmelweis, a Hungarian, Lister, an Englishman, Pasteur, a Frenchman, and Koch, a German, all making contributions of inestimable value to mankind.

This is a book alike for the layman and the doctor.

E. Falkner Hill

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